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INCIDENCE OF DENTAL CARIES AMONG MAYA AND NAVAJO INDIANS

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For nearly ten years one of the authors (Steggerda) has been making an anthropological study of various races. This paper deals with only the data concerning caries in teeth of Maya and Navajo Indians, as compared with people in a Dutch community (Holland, Mich.) and people on the island of Jamaica. During this study, the teeth in 5753 mouths were examined. The statistical evidence presented here includes only the five-year groupings of these people at the ages of 6 to 35. Detailed charts, to be published in reports of the Carnegie Institution of Washington, will include minor groups of Tuskegee Negroes, Pueblo Indians, and hybrid children from the city of Merida, Yucatan.

The examinations were made without instrumentation, and record only cavities that were visible with the aid of a tongue depressor. It is recognized that these figures do not represent the full amount of caries. But inasmuch as all examinations were made in the same manner and by the same examiner, and as this is a comparative study, their relative values should be comparable. All teeth missing from adult mouths were counted as carious. It is probable that some of these were lost by accident, or from diseases involving surrounding tissue; but the number was small and the error involved would be correspondingly slight. Deciduous and permanent teeth are placed in separate groups. All graphs are for the totals obtained. Graphs showing division of males and females, and yearly groupings, will be presented later. A slightly greater percentage of females have dental caries in all groups; also the average number of cavities per person having caries is greater in the females of all groups except the Navajo, where it is slightly less.

This paper deals with the (a) percentage of people having caries at various ages; (b) percentage of teeth involved; (c) distribution of occurrence of caries. *Fig. 1* shows the percentage of children having caries of *deciduous* teeth. This group is comprised of 1082 Navajos, 707 Mayas, and 970 Dutch, between the ages of 2 and 14. In the two Indian races, a few more than half are afflicted with caries of deciduous teeth. This contrasts sharply with the 90 percent afflicted in the corresponding Dutch group. Not only do more Dutch have caries, but the number of cavities per mouth is greater. The average number of cavities in deciduous teeth per mouth having caries for all ages is:

TABLE 1

Five-year groupings of indicated races, showing percentage of population having caries, percentage of teeth affected, and average number of cavities per mouth

AGE	DUTCH (WHITE)				MAYA (INDIAN)				NAVAJO (INDIAN)				JAMAICA (COLORED)			
	Number examined	Percent having caries	Percent of carious teeth	Average cavities per person having caries	Number examined	Percent having caries	Percent of carious teeth	Average cavities per person having caries	Number examined	Percent having caries	Percent of carious teeth	Average cavities per person having caries	Number examined	Percent having caries	Percent of carious teeth	Average cavities per person having caries
6–10	702	46.9	4.2	2.4	375	3.2	0.2	1.8	647	5.3	0.3	1.6				
11–15	533	88.2	12.8	4.3	319	10.7	1.9	1.5	634	17.0	1.0	1.7	304	69.4	10.2	4.1
16–20	123	96.7	24.1	6.9	106	37.7	2.5	2.0	491	32.6	3.3	2.8	144	71.5	18.6	7.2
21–25	..	..	..	..	76	60.5	6.1	3.3	60	58.3	7.4	3.5	86	80.2	24.0	8.4
26–30	..	..	..	..	43	65.1	7.2	3.7	21	66.7	7.0	3.2	44	90.9	26.6	9.4
30–35	..	..	..	..	25	72.0	8.9	4.4	8	50.0	9.4	6.4	21	95.2	28.3	9.0

Dutch, 4.8; Navajo, 2.8; Maya, 2.3. *Fig. 2* shows the percentage of *deciduous* teeth that are carious at ages 3 to 14, the two Indian races having less than half the amount in the Dutch. It is evident from these graphs that, with loss of deciduous teeth, both percentage of individuals having caries and percentage of deciduous teeth decrease.

Table 1 is a compilation of data on the incidence of caries in *permanent* teeth—the percentages of individuals afflicted and teeth involved. Multiple caries in individual teeth is not taken into consideration. The table, compiled from data for the mouths of 1358 Dutch, 944 Mayas, 1861 Navajos, and 599 Jamaica colored, compares four widely separated races, which, in addition to racial differences,

have great variations of habit and diet. The data in the table emphasize the importance of the relationship of age to incidence of caries. The number of people afflicted and the percentage of teeth involved constantly increase with advancing age. It is essential, when caries in a race is considered, that age be regarded as an important factor. The Dutch group contains only individuals aged 6 to 20. It is probable that, if this series were extended to greater ages, there would be an increase in the number of people affected and, more particularly, in the percentage of teeth involved. The Jamaica group contains both negro and mulatto. In *fig. 3*—a graphic illustration of *table 1*—the percentages of people afflicted by dental caries are plotted according to race and age. The two Indian races show no significant differences. This is interesting because of the heavy carbohydrate diet of the Mayas and the heavy protein diet of the Navajos. In both races, more than 50 percent are free from dental caries until the age of 20. In the Dutch group more than half have dental caries in the permanent teeth by the age of 9; more than 90 percent by the age of 14. This agrees with the findings of Brekhus (1), who examined 10,445 students at the University of Minnesota at an average age of 18. He reported that 97.5 percent had caries—an average of 10+ cavities or fillings per mouth. The examination of the Dutch group of like age shows 98 percent having caries—and 9.5 cavities per individual affected.

In *fig. 4* are plotted, according to age and percentage, the carious teeth of these races. To this has been added the dotted line representing the results of a survey of the St. Louis schools by the dental society of that city (2). This has been added to compare the accuracy of our method with a similar survey by dentists. It is probable that the Dutch children of Holland, Mich., have as much caries as the school children of St. Louis, and that the small differences in percentages are due to ordinary variations in methods of examination. The average number of cavities per individual having caries is approximately the same for the Dutch and Jamaica groups. The lower percentage of carious teeth in the Jamaica colored is due to the smaller number of people affected. The incidence of caries in the Indian races is approximately the same, there being no outstanding difference either in number of people affected or average number of cavities per

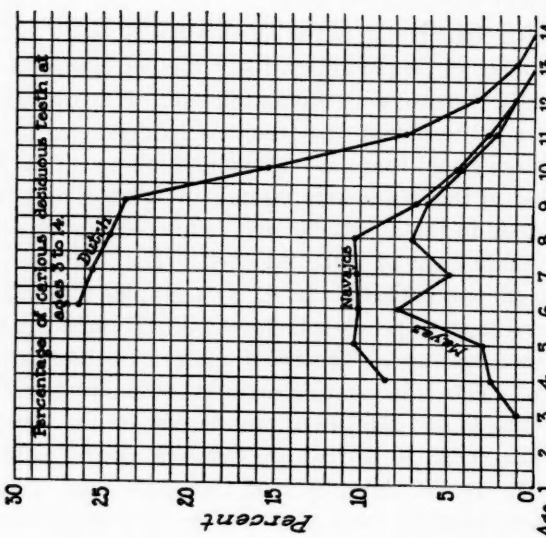

FIG. 2. Percentage of carious deciduous teeth in mouths of Maya, Navajo, and Dutch children; ages 3 to 14.

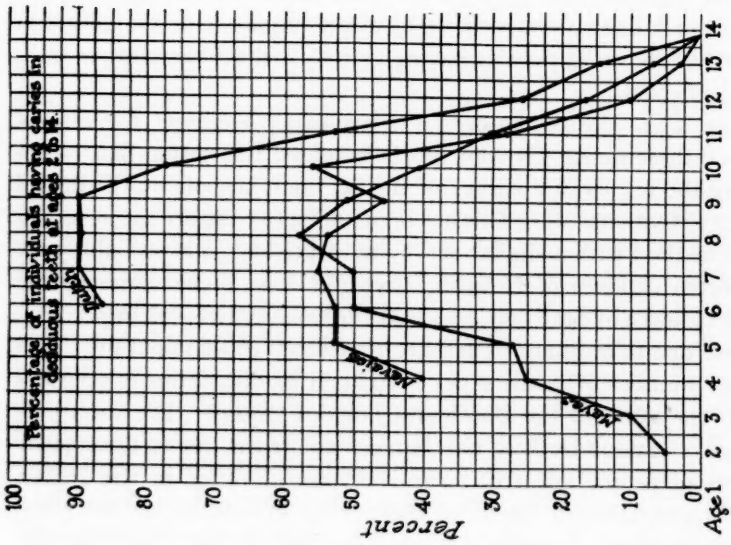

FIG. 1. Percentage of Maya, Navajo, and Dutch children having caries of deciduous teeth; plotted according to age.

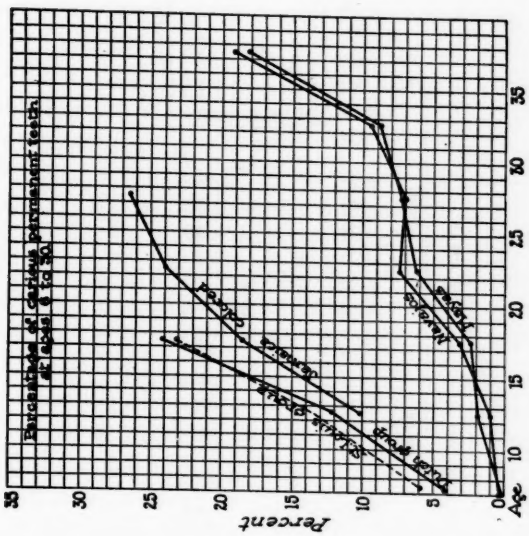

FIG. 4. Percentage of carious permanent teeth in mouths of Maya, Navajo, Dutch, and Jamaica colored; ages 6 to 40. (Dotted line represents percentage of carious teeth in St. Louis school children; from survey by St. Louis Dental Society.)

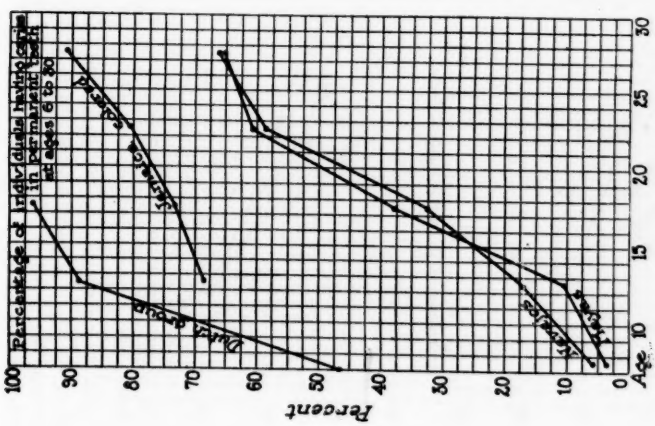

FIG. 3. Percentage of Maya, Navajo, Dutch, and Jamaica colored, having caries of permanent teeth; plotted according to age.

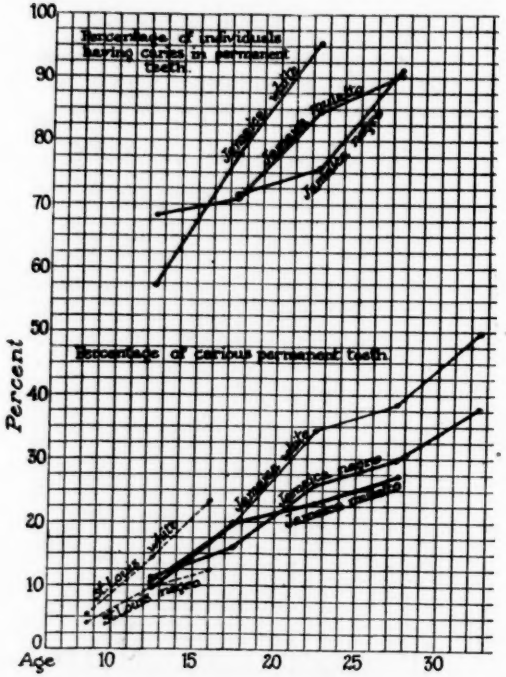

FIG. 5. Racial variation of people having caries; and percentage of teeth affected among races living in same communities. (Dotted line represents St. Louis children; from survey by St. Louis Dental Society.)

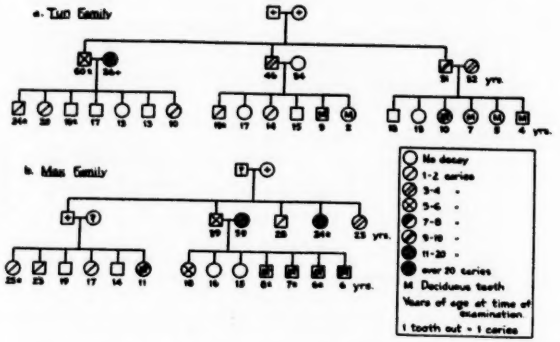

FIG. 6. Dental caries in two Maya families

person having caries. The remarkably low amount of caries among the Indians is worthy of further study. When a disclosing solution is applied to the teeth of Navajos, plaque formation is always found. No bacteriological work has been done to determine the number or even the presence of acid-forming organisms. Variations in the bacterial count of aciduric organisms and in the habits and diets might be discussed if the information were available. The possibility of racial resistance should also be given consideration. Analysis of a half-million examinations of first permanent molars, by Green (3) from data supplied by Wilson of the Cleveland Public Schools, showed that the time elapsing between eruption of the upper first permanent molars and decay of one-half of them is dependent upon racial stock and the economic group to which the individual belongs. Green stated that caries occurs earlier in the lower economic groups, but the greater variation of early caries is due to the racial stock. He presented evidence to show that upper first permanent molars of Negro, Italian, and Russian stocks, in the order named, last longer than those of other stocks.

In *fig. 5* is plotted the dental caries of inhabitants of Jamaica, both as to percentage of individuals having caries and percentage of teeth involved. This group was selected because habits and diets of the races living on this island are to a large degree comparable. The differences in percentage are not great, but it is significant that, in the white race, the percentage having caries is higher, and the percentage of carious teeth is greater. Figures taken from the survey of the school children of St. Louis show a similar difference between the white and negro races. Although economic variations in races living in the same community may alter diets and habits to some extent, the possibility of a racial-immunity factor should be considered.

In *fig. 6*, and in the appended summary, are shown the incidence of caries in two Maya families living as nearly as possible in the same environment. In these two families, of approximately the same age and number of individuals, the total number of carious cavities in one family is twice as great as in the other, suggesting the possibility that resistance to caries is inherited.

Families	Permanent teeth				Deciduous teeth			
	Adults		Caries		Children		Caries	
	no.	age	total	av.[1]	no.	age	total	av.[1]
Mex	13	23	61	4.7	5	7.6	27	5.4
Tun	19	24	40	2.1	6	6.2	5	0.8

Table 2 shows the distribution of caries in the previously discussed six groups. It represents ages from 6 to 25, with the exception of the Dutch—from 6 to 20. It refers to upper teeth, caries found, and location by percentage; *e.g.*, 62.8 percent of cavities in mouths of Navajos are in upper first molars, and 0.6 percent in central incisors. It does not indicate that 62.8 percent of upper first molars are carious. Only 4.8 percent of Navajo upper first molars are carious, but that represents 62.8 percent of all cavities found. Cavities among Navajos, in whose diet there is much protein and little carbohydrate, are

TABLE 2

Percentage distribution of caries in individual teeth, showing racial differences, without indicating percentage of teeth involved

	CENTRAL	LATERAL	CUSPID	1ST BICUSPID	2ND BICUSPID	1ST MOLAR	2ND MOLAR
Navajo	0.6	0.6	0.0	7.0	12.7	62.8	15.9
Maya	17.6	9.6	2.4	9.6	13.6	45.6	1.6
Jamaica White	14.8	15.1	2.6	15.3	15.9	27.3	9.4
Jamaica Mulatto	20.0	18.6	4.9	12.5	12.7	23.0	7.8
Jamaica Negro	19.3	19.6	6.7	14.7	9.5	18.2	12.3
Dutch	4.9	6.0	0.9	8.1	8.8	64.2	6.7

almost entirely confined to teeth having pits and fissures. The Mayas, who eat a large amount of carbohydrate, chiefly in corn, and a small amount of protein, have much more caries on teeth with smooth surfaces. Similarly, in the Jamaica group, where sugars are extensively used and the chewing of sugar-cane is a common habit—particularly among workers in the field—there is very much caries of anteriors. This possible relationship of caries and carbohydrate diet supports Bunting's contention that such diets favor presence of aciduric organisms as the important factor in the occurrence of caries.

[1] Average per person.

Other authors—Lipschitz (4), Mummery (5), Ottofy (6), Macphee (7–8), Shaw (9), Ferguson (10), Price (11), and Waugh (12)—have reported the incidence of caries in various races. No attempt has been made to correlate or compare this work with theirs because this survey is based upon age classification and differentiation of deciduous and permanent teeth. All these authors present evidence that primitive races have a lower incidence of dental caries.

Conclusions. 1. Navajo and Maya Indians have a low incidence of dental caries. The percentage of persons affected is small. This applies to both deciduous and permanent teeth.

2. Incidence of caries in the Dutch and Jamaica groups is high, both as to number of persons affected and percentage of teeth involved.

3. The amount of caries in any group of people is dependent, in large measure, on the age of the persons examined.

4. The findings suggest that, to a certain extent, resistance to dental caries may be a racial character.

5. There is a definite correlation between kind of diet and location of caries. Cavities on smooth surfaces of teeth are more common in races having high-carbohydrate diets.

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PHOSPHATASE IN HUMAN TEETH

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Phosphatase, an enzyme that separates phosphate from various organic combinations and thus facilitates utilization of phosphates in tissues, has been found in teeth of rats, rabbits, and guinea pigs (1) in amounts comparable to those in bone. Recently, its presence in human teeth was reported (2), the values indicating that the contained amounts of enzyme were very small. The present study was undertaken to obtain additional evidence regarding content of phosphatase in human teeth. The results lead to the conclusion that the contained amount is too small to be measured quantitatively by available methods. The differences observed by Provissionato among normal, carious, and pyorrhetic teeth could not be confirmed. Presence of an appreciable amount of phosphatase in a slightly calcified tooth-bud of a full-term human fetus was noted by Gross (3), indicating that the enzyme exists in human teeth during their formative stages.

Fresh human teeth were freed from all adherent tissue; crushed into very fine particles in an iron mortar; weighed; placed in water equal to 5, 10, or 20 times the weight of the sample, with a few drops of chloroform, and allowed to stand in stoppered flasks with occasional shaking at room temperature for 2 to 11 days; filtered through ordinary filter paper; and the filtrate placed in a refrigerator pending examination. The determinations were usually made within 12 hours after extraction was completed. It has been shown that such filtrates retain their activity for at least 2 weeks, when kept in a refrigerator. Phosphatase activity was determined by the methods of Jenner and Kay (4), and Bakwin and Bodansky (5). Both methods are based on the amount of inorganic phosphate formed by hydrolysis of β-glycerophosphate by the enzyme. In some of the experiments both the

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amount of extract used and the period of incubation were varied. The amount of liberated inorganic phosphate was determined by the method of Fiske and Subbarow (6).

Mixtures of 0.25 cc., 0.5 cc., or 2 cc. of tooth extract and 5 cc. of buffered substrate were incubated at 38°C. To the zero-hour tube, 2 cc. of 10 percent trichloracetic acid were added immediately. To the incubated tubes, the acid was added at the end of the incubation period. Although no visible precipitate of protein formed upon addition of the acid, the solutions were filtered. Of the filtrate, 5 cc. was used for the determination of inorganic phosphate.

A few values for phosphatase activity, using larger amounts of tissue extract, were observed in which turbidity set in upon adding the molybdate reagent, which made it difficult to evaluate the degree of color developed by the reducing agent (aminonaphtholsulfonic acid). This turbidity occurred with either substrate, and was caused apparently by some other substance in the tooth extract. In these cases small aliquots of extract were used.

The very small amounts of phosphatase phosphate were almost all of such magnitude as to be well within the limits of error of the method for the estimation of phosphate. Therefore the values obtained are not significant. However, inasmuch as the observed differences were almost all positive, the indication exists that phosphatase was present although in amounts so small that the available methods were not sufficiently sensitive to demonstrate it quantitatively. Moreover, except for one experiment, no relation could be shown between the amounts of phosphate liberated in 1 hour, 2 hours, and 6 hours, such as necessarily would be true if the reaction were due to enzymic activity. This result may also be due to inadequacy of the method.

Provissionato obtained values averaging around 0.22 mgm. of P, liberated in 6 hours per gm. of *normal* tooth; 0.73 mgm. of P, liberated in 6 hours per gm. of *carious* tooth; and 0.32 mgm. of P, liberated in 6 hours per gm. of *pyorrhetic* tooth. Efforts to duplicate his results were unsuccessful. In some experiments, his procedure was followed exactly, with the exception that aminonaphtholsulfonic acid was used as reducing agent instead of stannous chloride. This substitution cannot account for the observed difference in results.

TABLE 1

Data on tests for phosphatase in human teeth

TOOTH[1] CONDITION	EXTRACTION[1]		INCUBATION PERIOD: VARIABLE			INCUBATION PERIOD: CONSTANT		EXTRACT AMOUNT USED
	Water vol. per gram of tooth	Period	Difference between zero-hr. tube and incubated tube	Incubation period	P liberated per gram of tooth	Difference between zero-hr. tube and incubated tube	P liberated per gram of tooth in 6 hours	
	cc.	*days*	*mgm. P*	*hrs.*	*mgm.*	*mgm. P*	*mgm.*	*cc.*
1. Normal	10	7	+0.0034	2	0.041	...	...	1.5
2. Normal	20	2.5	+0.0035	24	0.430	+0.0013	0.157	0.25
						+0.0017	0.220	
						+0.0031	0.188	0.5
3. Normal	20	2.5	0.0	24	None	+0.0008	0.096	0.25
						+0.0010	0.121	
						+0.0064	0.115	2.0
4. Normal	20	2.5	...	..	...	+0.0003	0.018	0.5
						+0.0005	0.030	
5. Carious (9)[2]	5	11	+0.0070	1	0.042	...	...	1.5
6. Carious (3)	10	7	+0.0040	3	0.048	...	...	1.5
7. Carious (3)	10	7	0.0	2	None	...	...	1.5
8. Carious (2)	5	10	+0.0063	4	0.038	...	...	1.5
9. Carious	20	2	+0.0006	24	0.072	+0.0006	0.072	0.25
						+0.0001	0.012	
						+0.0004	0.024	0.5
10. Carious	20	2	0.0	24	None	+0.0002	0.024	0.25
						+0.0004	0.048	
						0.0	None	0.5
11. Carious	20	2.5	...	..	...	+0.0051	0.092	2.0
						+0.0054	0.097	
	50	2.5	...	..	...	+0.0034	0.153	2.0
						+0.0034	0.153	
12. Pyorrhetic (3)	10	7	+0.0050	2	0.063	...	...	1.5
13. Pyorrhetic	5	10	0.0	2	None	...	...	1.5
14. Deciduous: normal	20	2.5	...	..	...	+0.0018	0.108	0.5
15. Deciduous: carious	20	2.5	...	..	...	+0.0010	0.066	0.5
						+0.0016	0.096	
Rat incisor	100	1.5	+0.079	3	57.5	+0.126	86.8	0.5
Rat femur	50	1.5	...	..	...	+0.336	50.5	0.5

[1] The weights of permanent teeth, used in the extractions, varied between 0.86 and 2.95 grams; of deciduous teeth, between .06 and 0.49 gram. For the teeth indicated in items 5–8 and 12, the weights were greater.

[2] All debris and soft matter were removed from the cavities in carious teeth before use.

The author acknowledges her indebtedness to Professors William J. Gies, H. T. Clarke, and Maxwell Karshan for their generous interest and advice.

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SALIVARY AMMONIA AND ITS RELATION TO DENTAL CARIES[1]

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The properties of ammonia, and the fact that it is present in appreciable amounts in saliva, suggested an attempt to elucidate its function and possible relation to dental caries. Until Dodds (1925) reported on the chemical composition of saliva, as related to the etiology of dental caries, no attempt had been made to correlate ammonia content with this common disease. Dodds went only so far as to find that the ammonia of saliva was high as compared to that of blood, and that variations in proportions of ammonia and several other substances are so great that no clinical significance can be ascribed to the figures, at least for normal individuals. He did not extend the research to the analysis of salivas from individuals having caries, although it probably occurred in some of his normal subjects. Frank (1929) investigated salivary ammonia in reference to tartar formation. He did not refer to dental caries except to state that the ammonia may arise from products of local mouth disease. During the course of this research, Grove and Grove (1934, 1935) published papers in which they drew the conclusion that salivary ammonia is very intimately associated with dental caries. They believe that, as a solvent for mucin and plaques, it is an immunizing factor. The present author's research was initiated to obtain more data on salivas of normal individuals under various conditions, to analyze salivas of persons having caries, and to compare the results, in an effort to determine whether ammonia is a factor in the disease.

HISTORICAL. Pavlov (1888), not satisfied with Haidenhain's conclusions that protracted secretion produced marked histological changes in salivary glands and caused definite decrease in concentration of their organic solids, made quantitative determinations of total nitrogen (N) of saliva during rest and activity. During secretion, N substances appeared

[1] On the program of the fourteenth general meeting of the International Association for Dental Research, Louisville, Ky., March 14–15, 1936; *J. Den. Res.*, 1935–36, *15*, 373.

in the saliva, and simultaneously there was decrease of N in the secreting gland. The N loss in the gland, however, was much less than the N increase in the saliva. Evidently the gland received a partial replacement, which was more than half the total output. Others confirmed Pavlov's conclusions. Henderson (1900), investigating with greater care the total N in the secreting gland, found that equilibrium was established after loss during the period of activity. Wurster (1889) appears to have been the first to determine accurately the concentration of ammonia in saliva. He concluded that salivary "superoxid" changes ammonia to nitrous and nitric acids. He gave results for only two cases, in which the ammonia N amounted to 11.15 and 15.58 mgm. per 100 cc. of saliva. LeRoy (1908) described a rough quantitative test for salivary ammonia, stressing the clinical aspects of his results, but they are devoid of value, and their clinical significance was exaggerated.

Anrep (1920) investigated the distribution of N between mucin and non-mucinous substances of saliva. From a study of both active and resting glands he concluded: "The results show that within the limits of experimental error, the mucin of the saliva comes entirely from the mucin or mucinogen pre-stored in the gland; that the non-mucin nitrogen is derived from the body fluids, and that no formation of mucin or increase of nitrogenous cell-substance takes place during chorda stimulation." Hench and Aldrich (1922), in determinations of ammonia and urea in saliva, observed that ammonia increases at the expense of urea. They found 6 to 13 mgm. of urea-N plus ammonia-N per 100 cc. of saliva. In foul mouths the amount of ammonia was relatively great. Morris and Jersey (1923) reported that ammonia content ranged from 6.8 to 12.2 mgm. per 100 cc. of saliva. Their work was concerned mainly with uric acid, only incidentally with ammonia. Ammonia values were less regular than those for the other substances, indicating probably that ammonia is not a direct product of the glands. Possibly it is formed, after the secretion leaves the glands, through hydrolysis of urea. Dodd's investigation is especially interesting in this connection. With six other salivary constituents, he determined ammonia and drew attention to its high content in saliva as compared to blood. His ammonia-N values ranged between 1.5 and 13.4 mgm. per 100 cc. of saliva. He stated that the variations in composition of saliva are so great that no clinical significance can be given to the results, at least for normal individuals.

Clark and Shell (1927) studied the influence of diet on the inorganic constituents of saliva by making determinations of total- and ammonia-N on five prison subjects. They concluded that ammonia is definitely not an important factor in the reaction of saliva. Vladisco (1928), and Vladisco and Popesco (1929), investigated urea and ammonium salts in salivas of man, horse, dog, cat, and fox, and urea in the blood of man and the same animals. In salivas of 18 persons the maximum value for ammonia-N was 25.2 mgm.—the minimum, 3.0—per 100 cc. For the animals, the values were much lower: 1.5 to 0.5 mgm. in salivas of five horses; for a cat, 1.0 mgm. per 100 cc. However, they stated that saliva of the animals could not be obtained without pilocarpine or arecoline, and the values are thus probably low because of abnormal dilution. They made no analyses for alkali reserve, but nevertheless considered the rôle of the salivary glands in its maintenance. To explain the appearance of ammonia in saliva, two hypotheses were offered: (*a*) selective secretion of ammonia by the gland; and (*b*) transformation of urea into ammonia under the influence of the gland. The first seemed less tenable because the amount of ammonia in the blood is small. The second would liken the gland to the kidneys in preserving the alkali reserve of the organism. They concluded that elimination of ammonium salts indicates neutralization of a portion of the metabolic acids at the level of the salivary glands. Ammonia results from hydrolysis of urea. In this way, the fixed base is conserved.

Frank's paper (1929), on an extensive study of salivary ammonia with reference to its effect on tartar formation, is especially interesting, because it presents an historical review on salivary ammonia, and his experimental work is very closely connected with the problem of dental caries. Inspired air, in early investigations of the sources of salivary ammonia, did not account for it. Expired air, from lungs, mucous membranes of respiratory passages, or stomach, was also without effect. To Frank the only remaining possible sources were blood, tissue fluids, products of local mouth-diseases, and products of decomposition of N substances in saliva itself. He did not discuss these sources, nor himself study the origin of ammonia, but investigated its relation to tartar formation. In determinations of both free and combined salivary ammonia, he found a maximum of 17.0 mgm., and a minimum of 4.7, per 100 cc. of saliva in healthy individuals. These are total-ammonia values. In a case of lumbago, a value of 19.3 mgm. per 100 cc. was obtained. In a number of common diseases the values did not deviate from his normal values. Ammonia was most constant two to three hours after meals. The greatest daily variation was found in those persons who had the greatest amount of tartar. Also when saliva was allowed to stand at room temperature, the ammonia content increased very appreciably. In his chief work—analyses of free and combined ammonia, in normal and tartar-forming salivas—he tabulated results from many salivas, showing on the average several milligrams-percent more of salivary ammonia in tartar cases than in normal cases having no tartar. He divided tartar cases into three classes, in the order of increasing tartar formation, and found ammonia content to run parallel. Also, in five cases in which he determined ammonia before and after removing tartar, there was an average decrease in four—from 13.1 to 10.8 mgm. per 100 cc. The free as well as combined ammonia diminished in about the same proportion. He concluded that the ammonia in saliva plays an important rôle in tartar formation. For nephritic cases Maupetit (1932) found salivary ammonia-N somewhat higher than for normals—8.7 to 33.0 mgm. per 100 cc. of saliva; for normals, 5.1 to 10.8.

Grove and Grove (1934, 1935) studied, with reference to dental caries, the urea, titratable acidity, alkali reserve, pH, and especially ammonia in saliva of many patients in dental practice. They also studied the solubility of mucin and dental plaques in ammonium hydroxide and its salts. Their studies extended over several years. They concluded that (1) salivary ammonia appears to be the immunizing factor in dental caries; (2) ammonia is a solvent for plaques and salivary mucin; (3) plaque formation is retarded by high salivary content of ammonia; (4) dental caries is retarded when salivary ammonia is high; (5) plaque formation is retarded by local use of a solution containing ammonia; (6) "alkaline" diets raise salivary ammonia; (7) acid-base balance is apparently an important factor in dental caries; and (8) saliva of caries-susceptibles is stringy and adherent because of low content of ammonia.

DETERMINATION OF AMMONIA N. *Special apparatus and reagents required:* One Liebig condenser; 6-inch tube, one end bent and flared to fit 50-cc. Erlenmeyer flask by ground-glass joint. One 5-cc. micro-burette. Ammonia-free filter paper (rinsed successively in water, alcohol, and ether; then dried and placed in wide-mouth container and stoppered). Solutions of trichloracetic acid (approx. 1 percent), brom-cresol-purple (indicator), alizarin-red (indicator), sodium hydroxide (approx. 1.0 *N*), hydrochloric acid (0.01 *N*). Paraffin oil. *Procedure:* 5 cc. of fresh saliva are pipetted into 7 cc. of 1 percent trichloracetic acid solution, mixed and, after five

minutes or longer, filtered. Five cc. of filtrate are transferred to the 50-cc. distilling flask, and the following added: about 6 cc. of water; 3 drops of brom-cresol purple; 1 drop of paraffin oil; several small, clean, dry pebbles (preferably silica); and enough sodium hydroxide solution to produce persistent purple color (pH 6.5–7.0). Connection is then made with the condenser, and distillation carried on by applying a micro-burner flame under the wire-gauze support until all ammonia has been distilled. It is received into a small bottle containing a drop or two of alizarin red, and into which projects the adapter and the micro-burette tip. The ammonia is best titrated with the 0.01 *N* hydrochloric acid in this way, as it comes over. The bent glass-tube adapter need not dip into the distillate. *Calculation:* 6.73 × cc. of 0.01 *N* hydrochloric acid required = mgm. ammonia N per 100 cc. of saliva.

For the *non-mucin N-determination*, 5 cc. of filtrate are transferred to a 200 × 25-mm. Pyrex test-tube, 1 cc. of digestion mixture and two or three small silica pebbles added, and heat applied to the upright tube with a micro-flame until decolorization occurs. After allowing to cool somewhat and diluting, the mixture is transferred to the 50-cc. distillation flask, and treated as for the ammonia determination.

EXPERIMENTAL. *Collection and preservation of saliva.* It is well known that saliva contains N substances other than ammonia, particularly urea, which hydrolyze to produce ammonia, while held in the mouth or on standing without a preservative after expectoration. Accordingly, all collections were made after thoroughly rinsing the mouth; and decomposition was stopped by mixing with 1 percent trichloracetic acid solution as soon as the volume was measured—the proportion was 5 cc. of saliva to 7 cc. of the acid solution. In most of this work, where a single sample of 5–6 cc. was to be collected, it was first received into an ordinary graduated centrifuge-tube and then exactly 5 cc. were pipetted into a half-ounce bottle containing 7 cc. of the acid solution. (A stock of such bottles with the acid solution had been previously prepared.) On mixing well, the mucin was precipitated; after five minutes or longer, the mixture was filtered. The ammonia did not increase, to a measurable extent, on standing for a day or longer before filtering.

That mucin precipitates best in very dilute solution was considered. Anrep (1920–21) took this into account, and diluted between 5 cc. and 25 cc. of saliva to 100 cc. As indicated above, our customary dilution was 5 cc. of saliva to 7 cc. of the acid solution. In the experiments where both non-mucin N and ammonia N were determined, 5 cc. were diluted to 100 cc. In

this greater dilution the subsequent filtration is more rapid, but the large volume required for distillation is not so satisfactory.

No paraffin or other substance was chewed to increase saliva secretion, except where indicated. Most persons are able to secrete 5 cc. of saliva in five minutes or less. A majority of Bendixen's (1932) subjects secreted 0.2 cc. per minute; from others he obtained as much as 8.3 cc. per minute. The writer can as a rule, without stimulation, collect 5 cc. in about three minutes. From a few subjects it was impossible to obtain several cc. of saliva in 15–30 minutes. The psychic factor is unusually effective at times. In one case no saliva was secreted when it was to be collected directly into a centrifuge tube; but when a drinking cup was substituted, secretion was prompt and abundant.

Ammonia N (and non-mucin N) in various salivas. The experimental work is indicated in the accompanying illustrations and tables, and only brief explanations need be given. The concentration of ammonia N in salivas of four normal adults, between 20 and 25 years, three of whom were members of our laboratory staff, was determined on salivas collected at various times of the day. Meals and living conditions were those to which these subjects were accustomed. No other food, candy, etc., was reported to have been eaten during the periods. *Fig. 1* indicates some of the results. *Fig. 2* shows the values for the writer's saliva on three different days under normal conditions. *Figs. 3, 4,* and *5* represent the results of experiments on the ingestion of sodium bicarbonate and ammonium chloride. *Fig. 6* indicates effects of different substances on ammonia N in the saliva of four adult individuals. Saliva was collected at the beginning of the experiment; and again immediately after 5 cc. of each of the solutions had been kept in the mouth until 5–6 cc. of saliva was secreted. In the smoking experiments, the mouths were rinsed after smoking had continued for five to ten minutes; then the collections were made. In *fig. 6,* increases in ammonia N are shown above the normal base-line for each subject; decreases, below. The normal base-line is the ammonia-N value for each subject just before the beginning of the experiment. The actual values are given at the bottom. Non-mucin N and ammonia N were determined in the salivas of two individuals over periods of two and three hours (*fig. 7*). The object was to cause secretion of as much saliva as possible during these periods, so that the salivary glands would be strained, if possible, in their capacity to produce the above constituents. The usual breakfasts were eaten several hours previously, but otherwise no food was taken during the experiments. *Fig. 8* represents experiments on salivas of two dental students ingesting normal, acid,

and alkaline diets. Saliva was collected daily at 10:30 a.m. and 3:30 p.m. Normal periods of three days each precede and follow the one-week periods

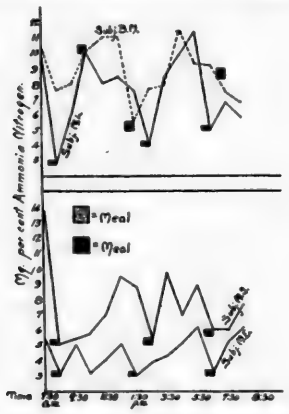

FIG. 1

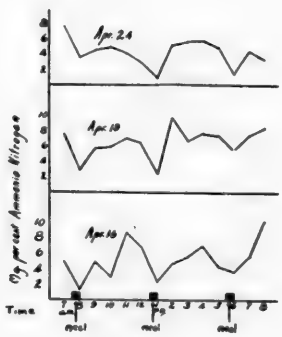

FIG. 2

FIG. 1. Ammonia nitrogen in saliva
FIG. 2. Ammonia nitrogen in saliva. Subject G. E. Y.

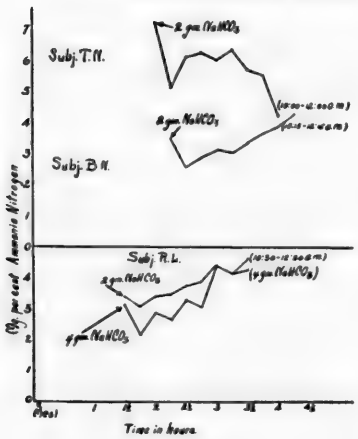

FIG. 3

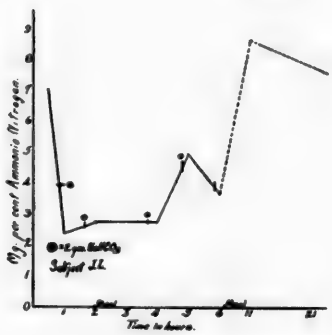

FIG. 4

FIG. 3. Effects of bicarbonate on ammonia nitrogen in saliva
FIG. 4. Effects of bicarbonate on ammonia nitrogen in saliva

on acid and alkaline diets. The normal diets consisted, as usual, of mixtures of acid- and base-forming foods in ordinary proportions. Daily

reports of foods eaten were made during the experiment. The acid diets consisted chiefly of proteins, with reductions in fruits and vegetables; the alkaline diets were reversed in these proportions. *Fig. 9* indicates results

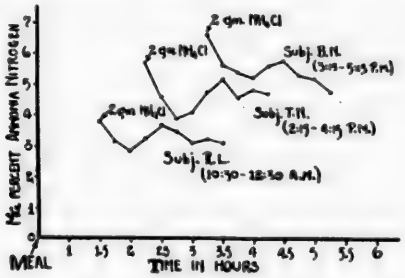

FIG. 5. Effects of ammonium chloride on ammonia nitrogen in saliva

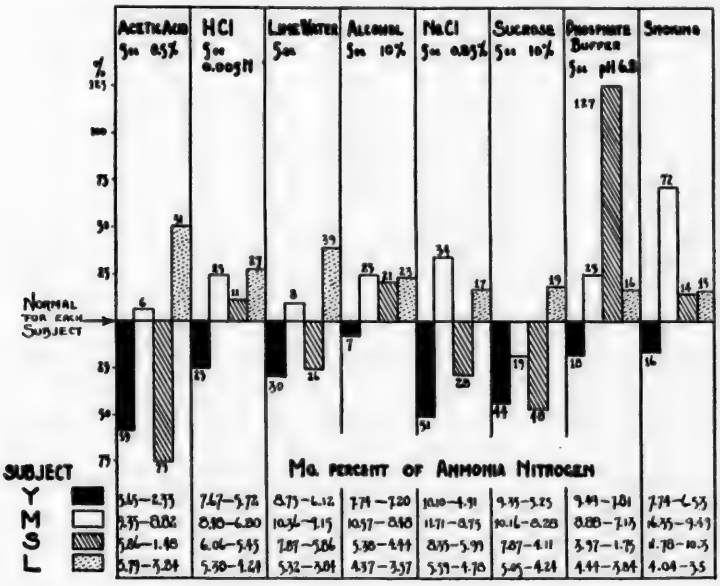

FIG. 6. Effects of different substances on ammonia nitrogen in saliva. Solutions kept in mouth until 5 cc. of saliva secreted.

on three out-patients, reporting for treatment for pernicious anemia, at Buffalo General Hospital. They had not secreted hydrochloric acid in the gastric juice for more than a year. Dr. Stuart Vaughan kindly selected

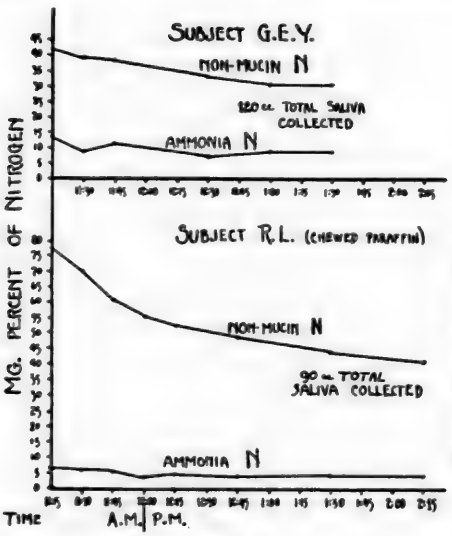

FIG. 7

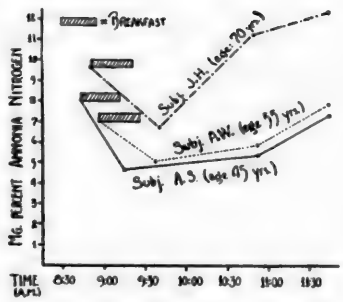

FIG. 9

FIG. 7. Non-mucin nitrogen and ammonia nitrogen in salivas of normal individuals. Saliva collected about every five minutes.

FIG. 9. Ammonia nitrogen in saliva of patients having pernicious anemia. No hydrochloric acid secreted in gastric juice for more than one year.

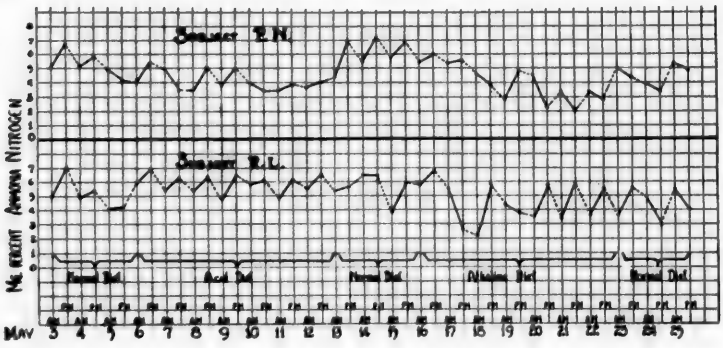

FIG. 8. Ammonia nitrogen in saliva under normal, acid, and alkaline diets

these individuals. Ammonia N in salivas of 45 subjects, all with definite dental caries, from the dental clinic at Buffalo City Hospital, was determined (*table 1*).

DISCUSSION. *Ammonia in saliva of normal individuals.* To obtain data on salivary-ammonia N of the average person, four subjects were chosen for collection of saliva on a number of occasions during most of the day (*fig. 1*). With one exception only, ammonia N diminished strikingly after meals. Recovery occurred rapidly. But

TABLE 1

Ammonia in saliva of individuals having dental caries

SUBJECTS		AMMONIA N	
No.	Average age	Average	Variations
	years	*mgm. percent*	*mgm. percent*
8	7–10	7.08	3.57–10.27
13	11–15	8.85	4.44–16.42
5	16–25	7.89	5.71–10.30
3	26–40	5.58	4.78– 6.19
11	41–60	10.02	5.38–16.96
5	61–76	9.99	6.12–14.81

TABLE 2

Salivary ammonia as found by different investigators

OBSERVERS	AMMONIA N	SUBJECTS
	mgm. per 100 cc.	
Hench and Aldrich (1922)	0.98–11.6	Normal
Morris and Jersey (1923)	6.8 –12.2	Normal
Dodds (1925)	1.5 –13.4	Normal
Clark and Shell (1927)	2.0 –26.0	Normal; prisoners
Vladesco (1928)	3.0 –25.2	Normal
Maupetit (1932)	5.1 –10.8	Normal
	8.7 –33.0	Nephritics
Grove and Grove (1935)	0.0 – 8.0	Caries-susceptible
	4.0 –10.0	Caries-immune
Youngburg (this research)	1.28–13.66[1]	Normal
	3.57–16.96	Active caries

[1] One individual gave a value of 16.35 mgm. per 100 cc. immediately after smoking.

factors other than ingestion of food also appeared to be effective, since there were decreases and increases between meals in some instances. The decreases are believed not to have been due to taking food, candy, or any other substance. To ascertain the variations in a single individual on different days, the writer examined his own saliva on three

different days (*fig. 2*). Again the effect of meals is striking. In every sample collected after a meal there was decrease, usually marked, in ammonia concentration. Considerable fluctuation from day to day is also apparent. These data and the values found for normal individuals, shown in other figures, indicate that there was always some ammonia in the saliva. As shown in *table 2*, values for normal individuals are between 1.28 and 13.66 mgm. of ammonia N per 100 cc. of saliva. This table also presents a summary of the recorded findings of other investigators. The minimum value of Hench and Aldrich (1922), 0.98 mgm. per 100 cc., is little less than our minimum value. Clark and Shell (1927) and Vladesco (1928) presented maximum values of ammonia N for normals that were about twice as great as any reported by others—26.0 and 25.2 mgm. per 100 cc., respectively. Evidently ammonia-free human saliva is very rare, if any such can be found. Grove and Grove (1934) are the only workers who have reported the absence of ammonia N from human saliva. Vladesco, however, reported "no ammonia" in a dog's saliva collected under pilocarpine stimulation. The values recorded by Grove and Grove can be accepted only as rough estimates when one considers the analytical processes they used. They extracted 2 cc. of saliva with permutit and diluted the colored solution to 200 cc. before estimating the color. This gives a very faint intensity; even with saliva containing 10 mgm. per 100 cc. of ammonia N, the intensity would be considerably below the standard for such quantitative determinations. It is likely that they missed the ammonia in some of their samples.

Although it is well known, from the work of Hench and Aldrich and others—and must be expected from theoretical considerations—that ammonia is formed in saliva by hydrolysis of urea, this is not a particularly rapid process. If saliva is promptly mixed with 1 percent trichloracetic acid solution, for example, there is no measurable decomposition. We found the usual amount of ammonia in saliva that had been secreted while holding 1 percent acetic acid in the mouth, or was immediately expectorated into 1 percent trichloracetic acid solution. Ammonia must be a regular salivary constituent. This is generally accepted. Whether ammonia is formed in the salivary glands, derived as such from the blood, or comes from inspired

or expired air, has been the subject of some investigation. Absorption from inspired or expired air apparently is not involved, according to several studies reviewed and summarized by Frank (1929). Since blood contains not more than 0.1 mgm. of ammonia N per 100 cc. and possibly none (Folin, 1922), it is also unlikely that salivary ammonia can be derived from blood. There remains only the possibility that it is formed in the salivary glands, perhaps in a manner similar to that of ammonia in the urine by the kidney. The amount of ammonia formed in the kidney is dependent on the quantity of fixed base available in the body, which in turn is usually dependent on the extent of acid production. If non-volatile acid-production is large, the base may be depleted enough to require ammonia formation, and the result is an increase of ammonium salts in the urine. If, on the other hand, fixed base is increased in the body by ingestion of alkali-yielding foods, such as fruits and vegetables, or by taking an alkali as such, *e.g.*, sodium bicarbonate, the acids formed in metabolism will be sufficiently neutralized without requiring formation of ammonia and the urine will be ammonia-free. This was first found by Dennis. If the same process takes place in the salivary glands, we should expect salivary ammonia to diminish after ingestion of bicarbonate. *Fig. 3* shows that salivary ammonia does diminish in such cases. In two subjects there were marked decreases after ingestion of 2 grams of bicarbonate; a third showed only a small decrease with 2 grams, but on taking 4 grams the decrease was marked. *Subject J. L.* showed a striking decrease on taking 10 grams of bicarbonate in the course of a day, in divided doses. But evidently these decreases cannot be ascribed to the acid-base equilibrium alone because similar decreases occur when ammonium chloride, an acid-producing substance *in vivo*, is ingested (*fig. 5*). After meals there is also nearly always a decrease, as previously noted.

In order to obtain more information on this mechanism, the effect of stimulating the salivary glands by holding various substances in the mouth was studied. *Fig. 6* indicates what was done and the results obtained. While *Subject Y* showed decrease in salivary ammonia with each reagent and on smoking, the results for the other subjects were on the whole somewhat irregular. No theory of the mechanism involved can be proposed. If ammonia is formed in the

cells of the glands, protracted secretion of saliva might tax their capacity to form ammonia and reduce its concentration. Anrep (1920–21) studied mucin- and non-mucin N from a similar viewpoint. He found that, on continual secretion of saliva, mucin diminished and finally disappeared. The non-mucin N followed a different course: it was not diminished, but continued at the same level throughout each experiment. Since ammonia N is part of the non-mucin N, it is likely that ammonia N was not diminished. Our own results of similar experiments on non-mucin N and ammonia N are presented in *fig. 7*. These N fractions were determined, in two subjects, for saliva secreted at a rather rapid rate. The results show that both non-mucin N and ammonia N diminished. In the salivas of *Subject R. L.*, non-mucin N fell from 77.8 to 41.7 mgm. per 100 cc., or 46.4 percent; ammonia N, from 7.1 to 3.2, or 55 percent. In *Subject G. E. Y.*, non-mucin N fell from 42.1 to 31.6, or 25 percent; ammonia N, from 13.1 to 9.3, or 29 percent. Thus, ammonia N diminished a little more rapidly than non-mucin N, although the difference is small. That they ran about parallel is significant. It is suggestive evidence that the same mechanism causes the fall in both ammonia and the other non-mucin substances. Since a fall in the main non-mucin N substances—urea, uric acid, creatinine and amino acids—cannot in this connection be involved in an acid-base equilibrium, neither can the ammonia be so considered from this evidence.

The results showing diminution in non-mucin N are apparently not in harmony with those obtained by Anrep on submaxillary saliva of dogs. He found a uniform level of such N. It does not appear that the saliva of his dogs was secreted slowly enough to allow replenishment of ammonia. Either there is a difference in metabolism of dog and man in this respect, or some other factor is responsible. Anrep analyzed submaxillary saliva; we studied mixed human saliva.

Ammonia in saliva of individuals having dental caries. If one attempts to find a causative factor in dental caries in the abnormal condition or composition of saliva *per se*, a number of well known constituents appear to require attention, among them mucin, ptyalin, maltase, urea, uric acid, creatinine, amino acids, ammonia, calcium salts, chlorides, sulfates, phosphates and carbonates—also such tran-

sient constituents as iodides—and the classical example, sulfocyanates. Of these constituents the following, especially, have been investigated in this relation: mucin, ptyalin, urea, ammonia, calcium, phosphates, carbonates, and sulfocyanates. Mucin has been considered from the standpoint of its mechanical and buffer effects; enzymes from their effects on production of sugars and ensuing formation of acids that attack the teeth; urea has been found to hydrolyze into ammonium carbonate which, with phosphates and carbonates, have been studied in connection with acidic and basic properties in relation to pH of saliva. The possible importance of pH has led to a number of investigations; but the evidence obtained in numerous analyses of human saliva by various workers has convinced nearly all that the pH of whole saliva cannot be the responsible factor. It is thought probable, however, that a localized low pH, at inaccessible points on the surfaces of teeth, may be the causative factor. In such cases low pH would be due to organic acids formed in the mouth by bacterial action on food debris.

Grove and Grove (1934, 1935) concluded that the concentration of salivary ammonia is determined by the acid-base balance in the body, which in turn is dependent on the kind of diet, and that salivary ammonia acts to prevent dental caries by dissolving mucin, which otherwise precipitates and forms plaques. They believe plaques form an adhesive contact for bacteria concerned in the attack on the teeth; if the ammonia N is less than 4.0 mgm. per 100 cc. of saliva, it is not of sufficient concentration; ammonia must be free, not in the form of its salts, to be effective. *Table 2* presents results obtained by us on salivas of 45 dental-caries subjects. It was not possible to correlate definitely ammonia N with age because the number of individuals of some ages was too small; but it seems that there is probably no variation with age. On the whole the data indicate that *the concentration of ammonia-N in salivas from persons who have active caries is about the same as that of non-carious or normal persons. This evidence tends strongly to show that caries is not dependent on ammonia concentration in saliva.*

These results are not in harmony with those of Grove and Grove. The values for ammonia N in caries-salivas, with one exception of 3.56 mgm. per 100 cc., are above the minimum of 4 mgm. per 100 cc.

set by Grove and Grove for caries-immune individuals, while a number of the values for normal salivas are below 4 mgm. per 100 cc. Even if it be agreed that caries is a transient process, it is not conceivable that all 45 subjects were inactive as to caries at the time their salivas were collected. Since these results differ from those of Grove and Grove, their theory that the production of ammonia in saliva depends on the acid-base balance of the body may be reëxamined. On this theory, if the body receives sufficient base through proper diet, there will be less need for ammonia, which is assumed to be present in the body, and thus the available ammonia will enrich the saliva and cause mucin to be kept in solution. This prevents caries. The increase in salivary ammonia after administration of base-forming foods —as stated by Grove and Grove—is the opposite of that which occurs in the body fluids. This we believe should call for close scrutiny before acceptance. And their statement that "the fluctuations of ammonia in other parts of the body, due to acid-base changes, being so well known," calls for explanation and criticism. Excepting the kidney, which manufactures ammonia that neutralizes acids and excretes resultant ammonium salts in the urine whenever fixed bases (Na, K, Ca, Mg) are insufficient, the tissues have not been found to form ammonia and thus to provide an ammonia store to be transported to another part of the body. The fixed bases of the tissues appear to be vitally necessary in the tissues to obviate ammonia formation. The small and questionable concentration of ammonia in the blood certainly does not come within the category of substances that function to neutralize acids. Whether the acid-base equilibrium can nevertheless influence the level of ammonia in saliva is questionable. The results in *figs. 3, 4, 5,* and *8,* in which an alkali ($NaHCO_3$) as such, and base-forming diets as well as acid-producing substances—ammonium chloride and potentially acid diets—were ingested, show not that salivary ammonia increased, but that it decreased. Our experiments were mostly for short periods, but perhaps long enough to be significant. Some of the experiments by Grove and Grove extended over very long periods; others were of short duration. They stated: "We have raised the salivary ammonia by the administration of sodium bicarbonate. The administration of acid-producing foods quickly lowers the ammonia of the saliva." They offer no data of

their own in connection with these statements excepting that they succeeded in raising salivary ammonia from 1 to 4 mgm. in a month by addition of orange juice to the diet. They cite reports of Jones, Larsen and Pritchard (1933) to the effect that natives of Hawaii on an alkaline diet were free from caries while those on cereal diets became very susceptible. As Koehne and Bunting (1934) state, however, "Jones, Larsen and Pritchard (1933) relate the dissolution of enamel so frequently seen in Hawaiian children and which they call odontoclasia, to present acidosis, but have presented no data on the alkaline reserve values for blood serum in support of their belief."

The further conclusion of Grove and Grove that salivary ammonia dissolves mucin and thus keeps it from forming plaques is also open to question. In view of the fact that one cannot determine the exact solubility of mucin in the usual way, the tests by Grove and Grove are well chosen and should be useful. In water mucin swells and forms a very viscid colloidal mixture or solution. More concentrated solutions, made by rendering a mixture of mucin and water somewhat alkaline, have no saturation-point but only become more and more viscous. In the saliva, mucin is believed to exist as potassium mucinate. Acidification of saliva causes flocculation of the mucin. Grove and Grove found different degrees of flocculation with various ammonium salts. They did not state the concentrations of these salts, which were doubtless much greater than any in saliva. Using only as much ammonia as occurs in most salivas—10 mgm. per 100 cc.—we found that there was no visible effect on mucin solubility; and 0.4 percent solutions of either ammonia or sodium hydroxide showed no difference in solvent action. We did not test the filtrates with any protein reagents because in very dilute solutions of proteins, particularly those having the properties of mucin, the sensitivity of these tests is unsatisfactory. While it is not doubted that ammonia and some of its alkaline salts have some power to dissolve mucin, our tests do not support Grove and Grove in their inference that the small amounts of ammonia in saliva are sufficient to prevent precipitation of mucin and even to dissolve mucin-containing plaques. Several other constituents of saliva affect the solubility of mucin; and it is logical to consider that, although no one of these may be sufficient, together they induce the solution of salivary mucin.

Opinion among dentists, so far as the writer can learn, is equivocal as to whether plaques are associated with immunity or increased liability to caries. Bibby (1935) states that there is no proof that the removal of plaques is of value in dental prophylaxis. He believes that they may be either advantageous or disadvantageous under different conditions.

Ammonia N of saliva in relation to "alkaline tide." In connection with the possibility that salivary ammonia may depend on the acid-base equilibrium of the body, and also because Bendixen (1932) found that there is a definite relation between salivary and gastric secretions in regard to volume and diastatic activity, it seemed desirable to determine whether the cause of "alkaline tide" (change in reaction of urine from normal acid to alkaline, following food intake) also induces change after meals in secretion of salivary ammonia. Examination of *figs. 1* and *2* does not indicate any relation between secretion of hydrochloric acid and salivary ammonia. Although decrease in ammonia nearly always follows a meal, the decrease is immediate and not prolonged; gastric secretion of hydrochloric acid, on the other hand, is more delayed and continues for a much longer period. Additional data are presented in *fig. 9*. As shown by Hubbard, Munford and Tyner (1933), individuals having anacidity—represented here by pernicious-anemia subjects—do not exhibit "alkaline tide." In our experiments, which covered periods of several hours after meals, concentration of salivary ammonia was not different from that of the normal subjects.

SUMMARY AND CONCLUSIONS. Variations in amount of ammonia in saliva of normal individuals at different times of the day, on different days, and under various conditions, were determined. Values ranging from 1.28 to 13.66 mgm. of ammonia N per 100 cc. of saliva under normal conditions were obtained. A value of 16.35 mgm. per 100 cc. was obtained for saliva of one individual immediately after smoking. Meals nearly always caused striking decrease in salivary ammonia. Bicarbonate ingestion also diminished it. The effect of ingesting ammonium chloride was neither striking nor constant; that of holding various stimulating substances in the mouth varied with different individuals. Changing from a normal to an acid- or an alkaline-diet, for a week, caused little change in ammonia content, although it tended to decrease on an alkaline diet.

The quantitative relationship between non-mucin N and ammonia N of saliva, during protracted secretion, was determined on two subjects; both N fractions diminished considerably in concentration and ran nearly parallel to each other.

Ammonia N was determined in saliva of forty-five individuals having active dental caries. Values ranging from 3.57 to 16.96 mgm. per 100 cc. of saliva were obtained. Comparing these with the values for normal non-carious salivas, there is no characteristic difference in ammonia content, and no indication that ammonia, *per se*, is a determining factor in susceptibility to dental caries.

The claim of Grove and Grove that low concentration of salivary ammonia, not exceeding 4 mgm. of ammonia N per 100 cc., is the main factor in susceptibility to caries was not substantiated.

Determinations of ammonia N in salivas of pernicious-anemia subjects, and of normal subjects, indicate that there is no effect related to the "alkaline tide."

The body mechanism that regulates concentration of ammonia in saliva has not been elucidated. Since ammonia concentration is definitely diminished by ingestion of bicarbonate, attention is directed to control by the acid-base equilibrium; other results, however, disagree.

The author appreciates the coöperation of all who aided in this work, special thanks being due to students H. G. Mickeleit, P. H. Newman, Robert Levine and John Leonard.

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STUDIES ON THE POTASSIUM AND CALCIUM CONTENTS OF HUMAN SALIVA[1]

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This work was undertaken to determine whether the ratio of K and Ca in saliva is related to immunity or susceptibility to dental caries. Part of the work was supplemented by correlation of the K:Ca ratio in blood. With the exception of one series of experiments, no effort was made to control or to make any change in the diet. The object was to determine conditions as they generally exist in the human mouth. Results were tabulated from 30 patients; 14 were decidedly active and susceptible; 10, immune; 5 had gingival erosion; 1 had acute ulcerative gingivitis. All borderline cases were excluded.

METHODS. *Saliva.*—Saliva was collected, without mechanical stimulus, in quantities from 12 cc. to almost 30 cc. (average, 15 cc.) at various times from 9 a.m. to 5 p.m. The saliva was filtered and then thoroughly shaken to insure uniformity of the filtrate. Determinations of Ca were made by the Clark-Collip (1) modification of the Kramer-Tisdall method (2). For K estimations, the saliva was completely ashed and K then determined by the Kramer-Tisdall method. *Blood.*—Fifteen cc. of blood, taken at the time saliva was collected, was allowed to clot and the clear serum poured off and centrifuged. K was determined by the Kerr (3) modification of the Kramer-Tisdall method; Ca, by the method used for saliva.

RESULTS. The analytical data show that the Ca and K values for "resting" saliva are subject to large variations in different individuals, and in the same individual. This observation agrees with the findings of Clark and Shell (4). From the data in *table 1* it will be observed that the average K content of saliva is 13 mgm. per 100 cc. lower in the active carious cases than in the immune ones, and

[1] Proceedings of eleventh general meeting of the International Association for Dental Research, Chicago, Ill., Mar. 18–19, 1933; *J. Den. Res.*, 1933, *13*, 183.

16 mgm. per 100 cc. lower than those for erosion. Since these differences only slightly exceed the limits of experimental error, no definite conclusions can be drawn until further observations are made.

In one experiment on a restricted diet, carried out on two very active cases and one immune, each patient ate the same kind of food in the same amount at the same hour each morning for eight days. Exactly one-half ounce of saliva was collected two hours after breakfast. During the first four days "resting" saliva was collected; on the suc-

TABLE 1

Ca and K contents of resting saliva

CONDITION ——→	IMMUNE	ACTIVE	EROSION
Number of patients	10	14	5
Number of determinations	53	35	9
Ca: mgm. per 100 cc.—average	6.9	6.3	7.2
—range	3.66–10.6	3.0–8.3	5.9–9.0
K: mgm. per 100 cc.—average	79	66.5	82.7
—range	22.0–127.0	31.0–109.0	67.0–95.0
K: Ca ratio (average)	11:1	10:1	11:1

TABLE 2

Ca and K contents of resting and paraffin-stimulated salivas compared in three subjects on restricted diet

		RESTING SALIVA					PARAFFIN STIMULATED				
						Av.					Av.
Active E. N.	Ca	6.7	6.2	6	7.2	6.5	5.4	5.4	5.7	5.3	5.45
	K	40	81.6	64	74	64.9	92	86	90	82.8	87.7
Active J. E. B.	Ca	6.7	6.7	7.7	5.6	6.6	7.4	7.2	6	8.3	7.24
	K	82	74	90	88	83.5	58	31	82	74	61.3
Inactive D. M.	Ca	5.4	6.5	6.8	7.5	6.55	7.2	5.4	5.2	4.8	5.65
	K	64	38	40	48	47.4	44	86	76	73	69.8

ceeding four days paraffin was used as a mechanical stimulus. The object was to determine, under these controlled conditions, whether resting and mechanically stimulated salivas differed in their contents of these elements. The data in *table 2* indicate that there was great variation in Ca and K values, in resting and stimulated salivas. These results do not show any relationship to immunity or activity. In the averages for all cases Ca was decreased, and K increased, by stimulation.

The following experiment was carried out to determine what percentage of K was combined with mucin (5). An excess of 95 percent alcohol was added to 5 cc. of saliva. The mucin thus precipitated was centrifuged and washed with 3 cc. of distilled water. Two drops of nitric acid were added, and the precipitate completely ashed. K was determined by the cobalti-nitrite method. In 14 experiments with salivas of different patients, K values ranged from 0.1 to 5.0 mgm. per 100 cc. of saliva, indicating that a very small percentage of K is combined with mucin. Therefore large mucin variations in saliva cannot be responsible for the great variation in amounts of K.

Part of this work on saliva was correlated by determinations of K and Ca in blood serum (*table 3*). Salivary K:Ca ratios of 10 patients, 4 of whom had active caries, varied from 4.5 to 25. The blood ratios in these patients ranged from 1.9 to 2.3. In the active cases blood Ca ranged from 9.5 to 13.3 mgm. per 100 cc.; K, from 20.4 to 30.0. The K:Ca ratio in saliva of six immune or completely arrested cases ranged from 5.6 to 17, again showing wide variation. The blood ratio ranged from 2.0 to 2.6. Blood Ca of the immune cases ranged from 10.0 to 10.9 mgm. per 100 cc.; blood K, from 20.2 to 23.1. These observations indicate that there is no relationship between these elements in saliva and blood serum in either immune or active cases, and agree with those of Clark and Shell (4), who reported the Ca and K contents for five subjects, all of whom were on known diets. For these they found that, per 100 cc., salivary Ca ranged from 4.6 to 11 mgm.; K, from 27 to 85. On the same subjects, in blood collected at the same time, Ca ranged from 7.4 to 11.8 mgm. per 100 cc. K values, determined in whole blood do not permit comparison, since red blood-cells were found to have a high K content. Clark and Shell quote Levine as obtaining values as low as 3.7 mgm. per 100 cc. for salivary Ca.

Hawkins (6), who has conducted a large number of Ca determinations on unfiltered saliva, reported a range of 6.0 to 110 mgm. per 100 cc. These values, confirmed by personal correspondence, are considerably higher than those in our determinations.

CONCLUSIONS. (1) Rather large variations occurred in the Ca and K contents of saliva, in the same patients at different times, and in different patients. (2) The K:Ca ratio averaged 10 to 1. No definite relationship of the K:Ca ratio to the occurrence of dental caries

TABLE 3

Correlations of K and Ca values in saliva and blood

PATIENT	SALIVA			BLOOD				ORAL CONDITION
	K	Ca	Ratio	K	Ca	Ratio	pH	
	mgm.	mgm.		mgm.	mgm.			
H. P.:	74.0	7.14	9.0	...	...	...	7.2	
age 16	69.0	6.30	10.0	23.4	12.02	1.9	7.5	
J. S.:	109.0	6.66	16.0	30.0	13.3	2.3	...	
age 18	32.0	7.1	4.5	25.2	11.8	2.0	...	Active caries
E. N.:	77.4	3.01	25.0	...	...	...	7.3	
age 16	36.0	5.0	7.0	...	...	...	...	
	54.0	5.5	9.0	20.4	9.3	2.1	6.4	
J. E.:	90.5	6.65	13.4	...	...	...	...	
age 30	86.0	7.6	11.3	21.7	10.3	2.1	7.45	
	93.0	8.2	11.3	20.2	10.3	2.0	7.46	
	88.5	5.07	17.0	...	...	...	7.4	
L. D.:	86.3	5.2	16.0	20.8	10.4	2.0	7.2	
age 25								
D. M.:	72.0	7.0	10.0	21.2	10.9	2.0	7.8	Immune; or not active caries
age 23								
J. C.:	105.7	7.9	13.4	26.0	10.6	2.6	6.1	
age 21	...	10.6	...	20.0	9.5	2.0	6.8	
	83.2	7.0	11.9	24.8	10.0	2.4	6.8	
	33.3	8.4	4.0	24.8	11.0	2.2	6.7	
	54.9	7.96	6.9	...	...	...	...	
J. B.:	89.5	8.36	10.0	...	...	...	...	
age 45	92.1	7.34	12.0	...	...	...	7.1	
	97.1	7.14	13.0	23.1	10.9	2.2	7.5	Erosion
	70.0	8.6	8.1	...	...	...	7.3	
S. C.:	96.0	7.0	14.0	17.96	10.62	1.7	...	
age 25	104.0	10.7	10.0	14.0	10.26	1.4	...	
	95.0	8.4	11.3	19.7	10.0	2.0	...	
	92.5	5.8	16.0	25.0	9.97	2.61	6.9	
	51.3	6.16	8.3	16.2	10.85	1.49	6.4	Acute ulcerative gingivitis
	58.6	4.3	13.6	24.28	11.9	2.04	...	
	57.8	4.8	12.0	20.14	11.33	1.78	6.8	
	69.1	6.4	10.8	21.08	12.28	1.7	6.8	
	80.2	9.0	9.0	22.36	11.25	1.98	...	
	41.08	4.6	9.0	32.5	10.0	3.25	...	
	54.5	3.7	14.7	23.5	10.12	2.32	...	

or erosion was found. (3) The K content of saliva of actively carious cases was slightly lower than for those of immune and erosion cases. (4) The Ca content in erosion cases was noticeably high. (5) While the blood K:Ca ratio remained fairly constant, the salivary ratio showed marked variation. (6) Only a small percentage of K occurred in combination with mucin; therefore large variations in the mucin content of saliva do not cause variations in K.

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HARDNESS TESTS ON TEETH[1]

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Hardness may be described as the resistance of one body to the "attempt" of another body to occupy the same space at the same time. Its exact magnitude depends upon the method by which it is measured. Two principles are in common use for determination of this property: a substance resists proportionately to its hardness (a) penetration by another harder substance, or (b) being scratched, or cut, by the sharp edge of another harder substance. Resistance to penetration as a method of determining hardness derives its importance from the present emphasis on metals and their properties. Resistance to scratch was developed for mineralogical use by Mohs, who arranged a hardness scale of ten minerals by placing one which would scratch another higher in his series.[3] Resistance to hammer blows and resistance to attrition are also used to measure hardness. Hardness tests on teeth have been made by Kopezky (1), Pickerill (2), Head (3), Proell and Schubert (4), Richter (5), Karlström (6), and others. Most of these investigators measured resistance of tooth tissue to a cutting edge; resistance to penetration was discarded for the most part as unsuited to hardness measurements on brittle, hard substances.

To find an instrument suitable for an extensive investigation of hardness of enamel and dentine, a number of standard hardness-testers have been employed. Several other hardness testers, not available in this laboratory, were used on enamel and dentine in laboratories of manufacturers of the instruments (page 278). The

[1] Proceedings of the International Association for Dental Research: *Journal of Dental Research*, 1933, *13*, 192; 1935–36, *15*, 156.

[2] Rockefeller Senior Fellow in Dentistry.

[3] The microhardness of the minerals comprising the Mohs scale has recently been estimated by Hodge and McKay: *American Mineralogist*, 1934, *19*, 161.

data obtained with these instruments on teeth were recorded to determine not only the hardness of dental tissues, but also the effect of the instruments on the enamel and dentine tested. The teeth used, chiefly incisors and cuspids, and a few molars and unerupted third molars, were taken at random from those extracted by Dr. W. R. J. Wallace, in Strong Memorial Hospital, and kept in 5 per cent formaldehyde in distilled water until used. The teeth had been extracted one day to several weeks before testing. No importance is attached to this lapse of time because qualitative data only were sought. Tests on enamel were made most frequently on the labial surface of incisors; tests on dentine, on teeth cut sagitally and smoothed on an aluminum wheel. For the sake of clarity, each instrument used, its principle, its effect on the tooth, and qualitative data obtained on enamel and dentine, together with data for steel and brass, will be presented separately.

Brinell Hardness Tester. The standard instrument in many industries, for determination of hardness of metals in production, is the Brinell Hardness Tester (*fig. 1*). A hard steel ball is pressed with a known load into the specimen to be tested, and hardness determined by measuring the impression. The *Firth Hardometer* (*fig. 2*) is a similar instrument, including a microscope for measuring the impression. The standard load for the Brinell is 3000 kilos (approximately 6600 lbs.). Teeth are unable to withstand such terrific pressure and (*fig. 3*) literally explode under a pressure of approximately 200 kilograms.

The *Rockwell Hardness Tester* (*fig. 4*) embodies the principle of the Brinell, but uses smaller loads and offers the alternative of a conical diamond or a steel ball for the impression. A pressure, through a lever system, of 100 kilos is used with the steel ball; 150 kilos, with the conical diamond. The steel ball in dentine gives the distortion shown in *fig. 5*; destructive effects of the conical diamond on enamel are shown in *fig. 6*. Data (*fig. 7*) were obtained with the Rockwell Hardness Tester on tempered steel,[4] anterior labial enamel, enamel of unerupted third molar, dentine, and brass. Steel and enamel show hardness of the same order of magnitude—roughly three times that of dentine and brass.

The *Monotron Hardness Tester* (*fig. 8*) gives (on one dial) the pressure necessary to push a steel ball a given, constant distance (shown on the

[4] Hardness values on steel and brass for the various instruments are taken from catalogs of their several manufacturers.

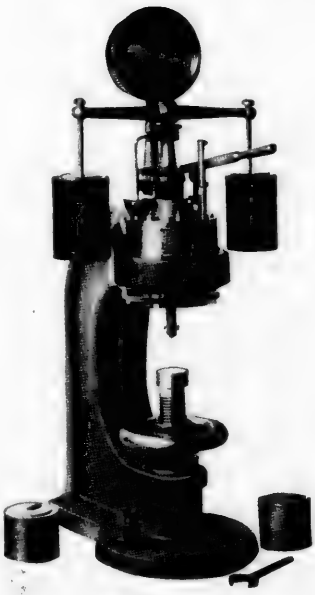

FIG. 1

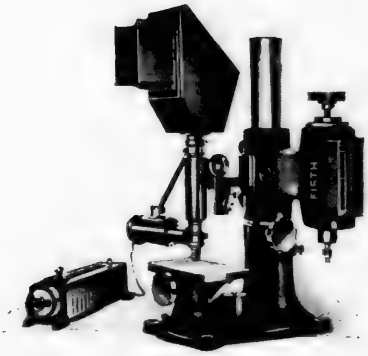

FIG. 2

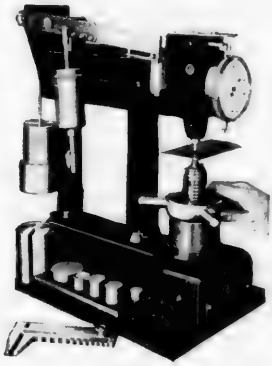

FIG. 4

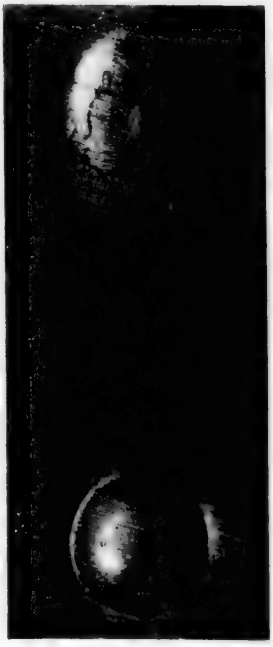

FIG. 5

FIG. 3a

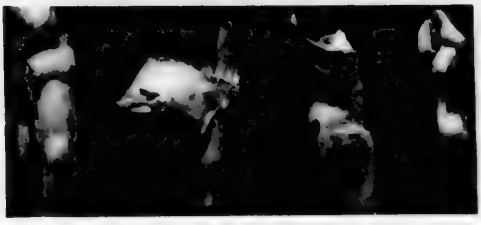

FIG. 3b

FIG. 1. Motor-driven hydraulic Brinell Hardness Tester. Load of 3,000 kilos applied through steel ball to test surface.

FIG. 2. Firth Hardometer (cut: courtesy of Tinius Olsen Testing Machine Co.). Designed to give direct Brinell readings (*fig. 1*) on thin or hard materials. Loads of 10 to 30 kilos applied, and resulting diameters of indentations read on screen of micro-projection head. Indenter head (here) swung away, and microscope stopped over impression for reading hardness.

FIG. 3. Tooth fragmentation resulting from use of Brinell Hardness Tester.

FIG. 4. Rockwell Hardness Tester: gives hardness numbers comparable to Brinell. Smaller loads impressed, using lever system on either steel ball (*fig. 5*) or diamond cone (*fig. 6*).

FIG. 5. Effect of steel ball of Rockwell Hardness Tester on dentine (×20).

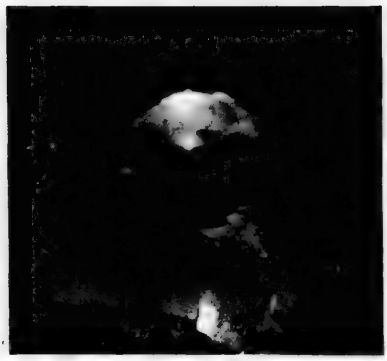

FIG. 6

FIG. 8

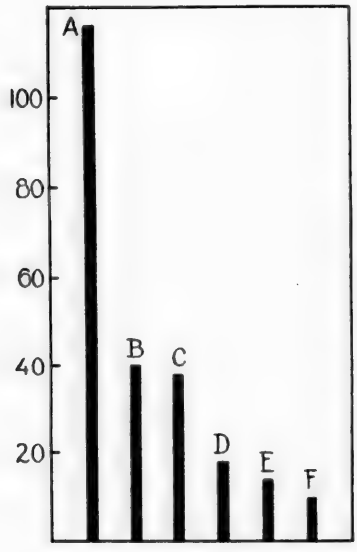

FIG. 10

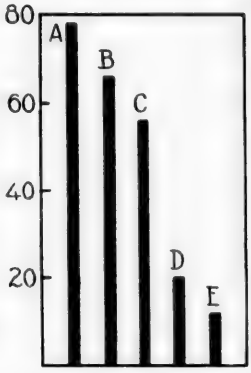

FIG. 7

FIG. 9

FIG. 6. Effect of diamond cone of Rockwell Hardness Tester on enamel (×20).

FIG. 7. Relative hardness of *A*, tempered steel; *B*, anterior labial-enamel; *C*, enamel of unerupted third-molar; *D*, dentine; *E*, brass—using steel ball of Rockwell Hardness Tester.

FIG. 8. Monotron Hardness Indicator (cut: courtesy of Shore Instrument and Manufacturing Co.). Larger dial (upper): pressure-gauge indicating pressure exerted by hand through lever arm. Smaller dial (lower): depth-gauge indicating depth of penetration of impresser-point for any pressure. Hardness values (*fig. 10*): pressures in kilos necessary to produce impression 0.045 mm. deep.

FIG. 9. Distortion produced by Monotron Hardness Tester on anterior labial-enamel (×20).

FIG. 10. Relative hardness of *A*, tempered tool-steel; *B*, artificial (porcelain) crowns; *C*, anterior labial-enamel; *D*, mild steel; *E*, brass; *F*, dentine—using Monotron Hardness Tester.

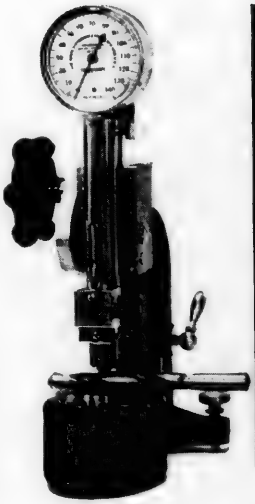

FIG. 11

FIG. 12

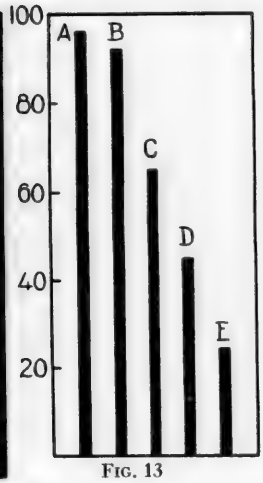

FIG. 13

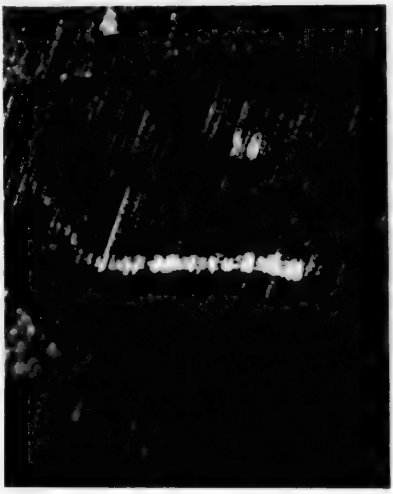

FIG. 15

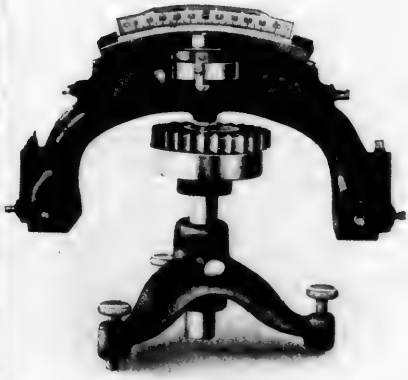

FIG. 14

FIG. 11. Shore Scleroscope (cut: courtesy of Shore Instrument and Manufacturing Co.). Hardness number (*fig. 13*): height of rebound of tiny hammer dropped from given height upon specimen.

FIG. 12. Effect of single impact of Shore Scleroscope hammer on anterior labial-enamel (×50).

FIG. 13. Relative hardness of *A*, tempered tool steel; *B*, anterior labial-enamel; *C*, enamel of unerupted third-molar; *D*, dentine; and *E*, brass—using Shore Scleroscope Hardness Tester.

FIG. 14. Herbert Pendulum Hardness Tester (cut: courtesy of Tinius Olsen Testing Machine Co.). "Time-hardness number" (*fig. 16*) obtained by measuring time in seconds for pendulum to make 10 swings, gently rocking. Ruby cone supports pendulum upon test surface.

FIG. 15. Tooth surface, roughly ground, marked by Herbert Pendulum Hardness Tester. White spot in dentine, above streak: mark of ruby cone (×20).

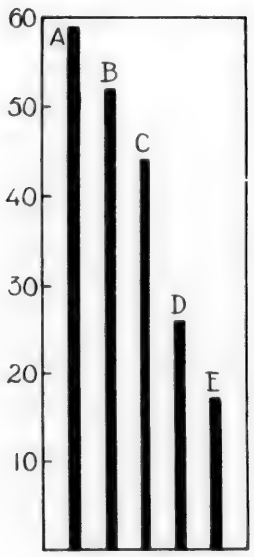

FIG. 16

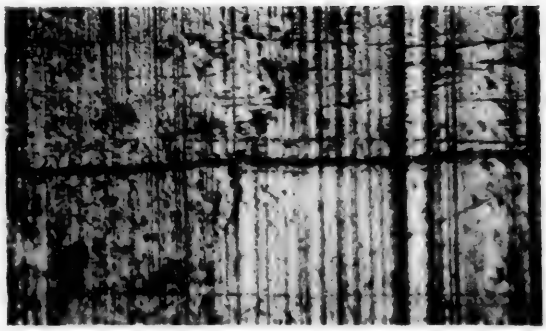

FIG. 18

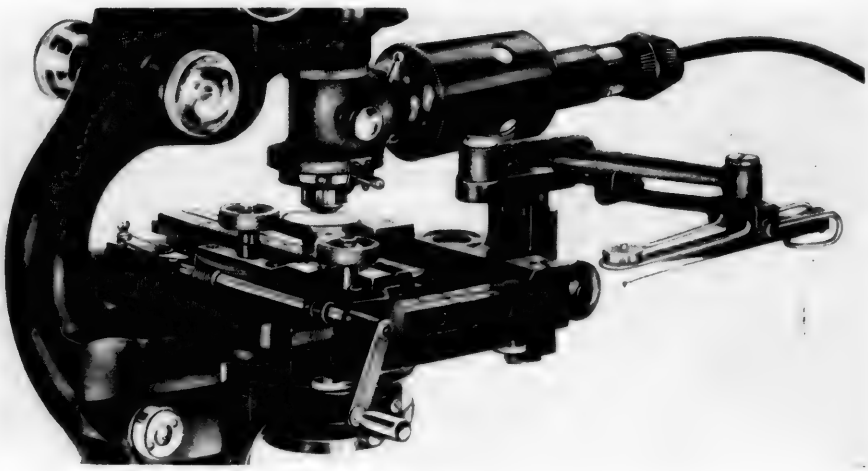

FIG. 17

FIG. 16. Relative hardness of *A*, steel; *B*, anterior labial-enamel; *C*, enamel of unerupted third molar; *D*, dentine; and *E*, brass using Herbert Pendulum Hardness Tester.

FIG. 17. Microcharacter (cut: courtesy of F. H. Bierbaum, Lumen Bearing Corp.). Tiny diamond, ground to corner of cube, cuts test surface under load acting through lever-spring system. "Micro-hardness" (*fig. 19*): squared width of "microcut" divided into 10.[4]

FIG. 18. Microcut crossing dentino-enamel junction. Enamel, left; dentine, right. Direction of cutting, right to left. Ridges in specimen: due to method of grinding surface Microcut in dentine: almost twice width of dark band (×430).

other dial) into the material being tested. The hardness is taken as the pressure in kilos necessary to push the steel ball $\frac{9}{5000}$ (0.0018 inch) $\frac{9}{200}$ (0.045 mm.) into the test piece. The effect of four tests on anterior labial enamel (*fig. 9*) may be described as destructive distortion. Data were obtained (*fig. 10*) with the Monotron for tempered tool-steel, artificial (porcelain) crowns, anterior labial enamel, mild steel, brass, and dentine. The hardness of anterior labial enamel is nearly half as great as that of hardened tool-steel, and roughly twice that of mild steel; dentine compares closely to brass.

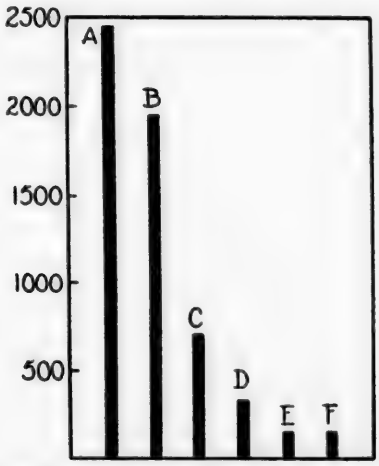

FIG. 19. Relative hardness of *A*, tempered steel; *B*, anterior labial-enamel; *C*, enamel of unerupted third-molar; *D*, dentine; *E*, amalgam filling; and *F*, brass—using Micro-character.

The *Shore Scleroscope Hardness Tester* (*fig. 11*) operates by dropping a tiny hammer on the surface to be tested, and measuring the height of its rebound. The effect of a single impact of the hammer is shown in *fig. 12*. Almost invariably the first rebounds were about half the height of later rebounds. A series of rebounds on anterior labial enamel were successively 40, 55, 80, 90, 87, 90. In hardness (*fig. 13*), tempered tool-steel, anterior labial-enamel, enamel of unerupted third molar, dentine, and brass varied in the order named, with anterior labial enamel almost equal to tempered steel.

Herbert Pendulum Hardness Tester. The penetration of a steel ball or

diamond point may be estimated in other ways than by direct observation. The Herbert Pendulum Hardness Tester (*fig. 14*) makes use of the speed of rocking. Essentially it consists of a weighted, inverted pendulum supported on a ball of steel or on a diamond or ruby cone. The time in seconds required for ten complete swings through a small arc is recorded as the time-hardness number. The effect (*fig. 15*) on a slightly smoothed surface of dentine is very superficial. The scratch occurred when the tooth slipped in the vice, allowing the ruby point to slide along the surface. This Tester indicates (*fig. 16*) that steel and anterior labial enamel have about the same order of hardness, while enamel of unerupted third molar and dentine lie between the values for the former and that of brass.

In *Microcharacter Hardness Tester* (*fig. 17*) the scratch principle is employed, under two fundamental conditions: the cutting point is ground with extreme precision to the corner of a cube; and the cutting point is mounted so that vertical pressure, exerted normal to the surface, is greater at all times than the horizontal pull applied to the point. The micro-cut (*fig. 18*) shows at a magnification of ×430 the effect of the cutting point on smoothed dentine. The data (*fig. 19*) obtained with the Microcharacter on tempered steel, anterior labial enamel, enamel of unerupted third molar, dentine, amalgam filling, and brass show that steel and anterior labial enamel have much greater hardness than dentine, amalgam and brass.

Summary. The hardness of tooth tissues was tested with Brinell, Rockwell, Monotron, Scleroscope, Herbert Pendulum, and Microcharacter hardness testers. In several cases the instruments caused destructive distortion of the material tested. Each of the instruments confirmed qualitatively the hardness data obtained with the others, although the values for hardness varied widely for the same tissue on different instruments.

The author acknowledges the indispensable aid given by Mr. W. F. Shore, Shore Instrument and Manufacturing Company (data on the Monotron, slides and electrotypes); Mr. Thorsten Y. Olsen, Tinius Olsen Machine Company (data on the Herbert Pendulum Hardness Tester, slides and electrotypes); Mr. C. H. Bierbaum and Mr. Harold McKay, Lumen Bearing Company (data on the Microcharacter, slides and electrotypes); the Engineering Department, University of Rochester (use of Rockwell Hardness Tester); Mechanics Institute (use of Brinell and Herbert Pendulum Hardness testers); Will Corporation (cuts and electrotypes); Eastman Kodak Company (use of

Shore Scleroscope and other instruments); and Dr. W. R. J. Wallace, Strong Memorial Hospital, and Drs. Van Huysen, Bibby and Dobbs, Rockefeller Fellows in Dentistry, School of Medicine and Dentistry, University of Rochester (technical assistance).

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DIETARY FACTORS IN RELATION TO MOTTLED ENAMEL[1]

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The presence of fluorine (F) in water supplies was definitely established in this laboratory as the cause of mottled enamel (1). In 1930, before this relation was known, the possibility that this dental defect was the result of dietary deficiencies had been investigated. A quantitative dietary study (2) was made of children who had mottled enamel. The food intakes of these children were then compared with those of children who had normal teeth, and all intakes were compared with dietary standards for optimum growth and nutrition. All deficiencies were considered in relation to presence or absence of mottled enamel. Special interest lay in possible deficiency of the tooth-forming elements, calcium (Ca) and phosphorus (P). Results showed, however, no significant differences in Ca, P, protein, or energy values of the diets of children in the mottled-tooth group as compared with children in the non-mottled-tooth group. In fact, the food intakes of all these children were remarkably optimal. It was observed and recorded, however, that the children with the severest form of mottled enamel—i.e., much pitting—had a much lower intake of vitamin C; and a possible significance of this fact was suggested. The conclusion drawn at that time, that the cause of mottled enamel must lie in some environmental factor and not in a dietary deficiency, was shortly confirmed by the finding of the causative relation of F in the water-supply of the afflicted persons. Variation in severity of the defect in human subjects has, however, been observed among those who use the same F-containing water-supply. The possibility of inhibiting the destructive decalcifying action of F by means of dietary improvement seemed worthy of investigation. It was logical to suppose that differences in nutritive condition might explain, at least in part, the ob-

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served differences in apparent susceptibility in children to the action of F. Many experiments were, therefore, carried out in this laboratory to determine the relation of diet to the action of F in the production of mottled enamel.

EXPERIMENTAL. The experiments described below have been carried out with young rats, taken at weaning (28 days) from the stock colony and reared on Sherman Diet B ($\frac{2}{3}$ ground whole wheat, $\frac{1}{2}$ whole milk powder, and salt equal to 2 percent of the weight of the wheat). Previous work in this laboratory (3) showed effects on incisors of F at definite percentage levels in stock rations. The first outwardly observable effect was produced in young rats when the stock ration contained 0.0014 percent of F (in NaF) or, more exactly, when 1 mgm. of F per kilo of body weight was given daily. Incisors became slightly bleached, and by means of a hand lens or low-power binocular microscope (magnification 10 ×), fine lines or striations of normally orange-pigmented enamel, alternating with lighter lines of bleached enamel, were observed. After dosage with higher concentrations of F, the effect on incisors became progressively more severe. At the 0.0113 percentage level of F, the teeth lost their luster and pigment, and became dull white and non-translucent to the naked eye. When the F level was increased to 0.0226 percent, pitting and chipping of enamel, and more pronounced chalky whitening, resulted. At the still higher F level of 0.0452 percent, the teeth were severely corroded and usually worn down to the gum line; further eruption practically ceased; and, in addition, development of the whole skeleton was strikingly retarded. In the investigations described below, F was incorporated in the experimental rations at levels of known influences on the teeth, and all possible remedial dietary effects were judged according to these criteria.

RESULTS. *Variations in Ca and P contents of the ration.* The teeth of rats that receive F contain lower percentages of ash than those of the litter-mate controls (14). F dosage in high concentrations lowers the ability of the animals to retain the tooth-forming elements, Ca and P, as indicated by their greater excretion of these elements (5). The possibility of nullifying the effect of F on calcification of the teeth, by increasing the Ca and P contents of the diet, singly or together, thus suggested itself. Accordingly, experimental rats were

placed on diets in which the amounts of Ca and P were varied within rather wide limits. In one series, the adequate stock-colony diet (Sherman's Diet B) was used as a base. Ca and P contents were increased as indicated in *table 1*—Ca:P ratios were varied widely by these additions. F was added, as NaF, at rising levels of 0.0113, 0.0226, and 0.0452 percent of F. In every case, control animals were given the same basal diet, with the corresponding mineral additions but without added F. Comparisons were made of the teeth of all animals at weekly intervals over a six-week period. In no case did the Ca or P addition to the ration prevent the action of F upon the teeth, nor was it possible to observe any lessening in the severity of

TABLE 1

Composition of diets with variable contents of Ca and P

ADDED F	ADDED MINERAL COMPOUND			
	Substance	Ca	P	Ca:P
percent		*percent (approx.)*		*ratio*
0.0	None	0.5	0.5	1.0
	$CaCO_3$	0.9	0.5	1.8
0.0113	$CaCO_3$	1.1	0.5	2.8
	$CaCO_3$	1.7	0.5	3.4
0.0226	$CaCO_3$	2.1	0.5	4.2
	$Ca_3(PO_4)_2$	1.3	0.9	1.4
0.0452	$Ca_3(PO_4)_2$	2.1	1.4	1.5
	K_2HPO_4	0.5	0.9	0.6
	K_2HPO_4	0.5	1.3	0.4

the defect produced. Mottled enamel resulted in spite of the fact that the diet was fortified with rather large amounts of Ca or P, or both. At the 0.0113 percent F-level, the incisors of the rats lost their luster and became characteristically dull and white in appearance. At the 0.0226 percent level, the enamel was typically pitted; and at the 0.0452 percent level, severe corrosion of the teeth resulted in each case, regardless of the fact that the ration contained excess of the mineral elements of which enamel is composed. At the two higher F-levels, addition of calcium carbonate, or lactate or phosphate, materially inhibited the growth-stunting effect of F, but the damage to the teeth was apparently not mitigated.

The question arose whether it might be possible to prevent the decalcifying effect of F, if F were given at a very low level, sufficient only to produce the mildest type of mottled enamel. Accordingly, the experiment was repeated, and Ca and P additions were made to the diet when F was at the minimum dietary concentration that previous experiments in this laboratory had shown produces a toxic effect upon the teeth—1 mgm. per kilo of rat per day, or approximately 0.0014 percent of F in the ration. Again it was noted that the Ca or P additions did not prevent the action of F. The teeth of both series of experimental animals showed fine lines of normal pigmentation alternating with abnormally bleached areas, characteristic of the minimum F-effect on rat incisors.

In order to determine if diets low or actually deficient in mineral elements make F even more toxic and destructive, or increase susceptibility to it, rats were given diets suboptimum in mineral contents. Rations markedly deficient in Ca or P were obtained by the use, in a synthetic diet, of Osborne and Mendel's salt mixture, in which either Ca or P salts were omitted or reduced in amount. In another series, a less drastic lowering of mineral content, which would be more typical of suboptimal diets of human beings, was secured by varying the stock ration in the following way: in the suboptimal-P ration, white flour replaced whole-wheat flour, reducing the P content of the ration from 0.5 to 0.3 percent, whereas the Ca content remained practically the same. It is recognized that this ration was suboptimum in other respects for long continued experiments. When the amount of whole milk in the stock ration was reduced from one-third to one-sixth, both Ca and P contents were lowered, but Ca was reduced to a greater degree than P. F was incorporated in these rations at a concentration of 0.0113 percent, and also at the minimal toxic concentration, 0.0014 percent. A control group was given the same suboptimal mineral rations without F; and another, the rations adequate in Ca and P with F. Comparisons of the incisors were made daily so that the time of appearance of typical F-effects, and their severity, could be recorded. Again, the results showed no direct relation between Ca and P contents of the diet and action of F. No difference in severity of dental defects was recorded, excepting cases in which the Ca or P content of the ration was so inadequate

that the mineral deficiency in itself interfered with normal dental calcification. It was noted that the incisors of rats on rations markedly deficient in Ca were dull and lusterless, whereas the low-P diet resulted in some bleaching of the pigment. Addition of F to these diets resulted in somewhat more severe dental damage, probably owing to what might be called an "additive" effect. However, it was not possible to produce typical F-damage with lower doses of F when the ration was suboptimal in either Ca or P. It can perhaps be concluded, therefore, that diets faulty in mineral make-up do not render an individual more susceptible to F, but the combination of F damage and that due to inadequate Ca and P intakes may make the teeth "doubly" inferior and defective in formation and calcification.

Variations in content of vitamin D in the ration. The possibility of preventing mottled enamel by insuring the presence of excess of vitamin D was investigated by making large additions of this vitamin to the rations of rats receiving F. Vitamin D was supplied in the form of cod-liver oil or 250 D viosterol. Cod-liver oil was incorporated in the adequate ration at the levels of 2 and 4 percent. Viosterol was given separately—1 drop daily. These vitamin-D additions were made to the stock-colony basal-ration; also to the diets that had been fortified with additional Ca and P. In no case was the destructive action of F prevented by enriching the diet with this vitamin. F produced characteristic interference with calcification of enamel, even in the presence of large surpluses of the calcifying vitamin.

Thirty children who were drinking water containing 2 to 5 parts per million of F were also given supplemental feeding of 10 D cod-liver oil, and 250 D viosterol, daily over a period of three years between the ages of 6 and 9. Periodic examinations of their teeth were made and compared with the teeth of a control group drinking the same water but not receiving the supplements of vitamin. In no case did these supplements prevent the appearance of mottled enamel upon the permanent teeth of these children as they erupted, nor was the resultant mottling less severe than that observed in the control group. We cannot agree, therefore, with the statement by Chaneles (6) that the action of F can be prevented by feeding cod-liver oil.

Variation in content of vitamin A in the ration. Many experiments have indicated that vitamin-A deficiency may be eliminated as a cause of mottled enamel. Other experiments have since been conducted to bring out any relationship of vitamin A to susceptibility to the toxic action of F. In one series, animals whose body reserves of vitamin A were exhausted, by deprivation of the vitamin over a four-week period, were placed on a ration suboptimum in vitamin A and containing 0.0113 percent of F. The incisors of these rats presented the dull white and mottled appearance shown by the control rats on the same ration fortified with additional vitamin A in the form of cod-liver oil. The suboptimal intake of vitamin A did not appear to increase the toxic effect of the F dosage. When, however, rats were placed upon purified ration fully adequate in all respects except that it was entirely deficient in vitamin A, the incisors became dull, blunt, white, and opaque, and ceased to grow (7). These dental changes always go hand in hand with other symptoms of vitamin-A deficiency. After addition of F to this ration, the teeth were increasingly defective and poorly calcified, but no direct relationship between F toxicity and vitamin-A deficiency could be seen. On the other hand, addition of large excesses of vitamin A to the stock ration, in the form of cod-liver oil, did not offset or delay the destruction caused by F. In every case there was F damage characteristic of the concentration of F in the ration.

Variation in content of vitamin C in the ration. Although the effect on the teeth of deficiency of vitamin C in the diet is quite different from F damage, an influence of one upon the other seemed possible. As stated previously, study of the diets of children having mottled enamel, caused by drinking water containing F, pointed to the fact that the group of children having the most defective teeth, with most severe pitting and corrosion, consumed less fruits and vegetables, which are the best sources of vitamin C. Young guinea pigs were therefore placed on a vitamin-C-free ration and given graded doses of tomato juice, varying in amount from 0.5 to 5 cc. daily. NaF was given by pipette into the mouth or by subcutaneous injection daily. These experiments did not show any relationship between vitamin-C intake and injury to dentition due to F. Animals on the smallest supplements of vitamin C became extremely scorbutic before typical signs of severe fluorosis were evident. F damage in all cases appeared

to be a function of the amount of F, and was not appreciably altered by consumption of vitamin C.

The guinea pig is not a thoroughly satisfactory animal for the study of F injury. It is impossible to see the initial changes in the teeth as early and as clearly as in the rat. Since rats probably synthesize sufficient vitamin C for their adequate nutrition, they could not be used to study the effect of inadequate vitamin C on F injury. However, daily feeding of excess of vitamin C to rats as orange juice (5 cc.) and as ascorbic acid (2.5 mgm.) did not prevent appearance of the typical bleaching effect of F when the minimum toxic dose of F (0.2 mgm. per day, 1 mgm. per kilo, or 0.0014 percent of the ration) was given; nor was the severity of the effect at the higher levels of F feeding lessened by the relatively high consumption of vitamin C.

Variation in content of protein in the ration. In one series of experiments, variation in the protein intake was secured by use of 6, 9, 12, or 18 percent of casein, in diets adequate in all other respects, and which contained F at 0.00625, 0.0113, and 0.0226 percentage levels. Growth rates of young rats on the lower protein levels were materially retarded, but in every case the F damage to the incisors was typical of the concentration of F, irrespective of the protein content. Suboptimal intake of protein did not result in any apparent difference in the effects of F.

To determine effects of F on protein digestion and metabolism, experiments were conducted in which the balances between intake and outgo of protein in 16 animals given F, and in 8 that did not receive F, were compared. The appended results show that the young animals that received F made use of the protein in their ration almost as efficiently as the litter-mate controls that did not receive F:

Added F in ration: percent	*Average coefficient of digestibility of protein*	*Average daily retention of nitrogen*	
		Mgm. per gm. of body weight	*Percent of intake*
0.0	89.3	0.305	29.7
0.0226	88.8	0.298	33.8
0.0452	89.5	0.262	29.2

Nor did F interfere with the digestion of proteins, although *in vitro* F has been observed to inhibit the action of certain proteases. The coefficient of digestibility of protein was the same for all the rats.

Variation in acid-base balance of the ration. The potential acidity or alkalinity of the stock ration was increased by addition of 6 percent ammonium chloride or sodium carbonate, respectively. NaF was then incorporated in these rations, which were potentially strongly acid or basic, at 0.0014, 0.0113, and 0.0226 percentage levels of added F, or given separately—1 mgm. per kilo of rat body-weight. Again it was noted that the F damage to incisors was remarkably constant for each F concentration, regardless of potential acidity or alkalinity of the basal ration.

Discussion. These experiments, conducted with approximately 1500 animals, indicate that mottled enamel does not result from a deficiency of any known dietary essential; also that such deficiency does not increase susceptibility to the toxic action of F in the production of mottled enamel. Variation in the severity of this characteristic dental defect appears to be a function of the intake of F.

It was not possible to prevent the destruction of dental tissue in rats, nor to lessen the severity characteristic of a certain F intake, by any of the dietary improvements investigated. F exerted its injurious effect to its maximum degree, regardless of other experimental conditions. It was observed, however, that teeth defective in formation as a result of dietary lack of Ca and P, or vitamin A or C, presented more severe damage; but the effect is believed to be an "additive" one. A dietary deficiency not severe enough in itself to show outward abnormal changes in rat incisors was without influence on injury due to F.

In no case did correction of a dietary deficiency prevent F injury, nor did large excesses of any dietary essential in the ration inhibit fluorosis.

It seems logical to believe, therefore, that observed differences in the severity of mottled enamel in human beings are almost entirely the result of differences in the amount of F ingested. The concentration of F in public water-supplies (8) is known to vary within rather wide limits. Concentrations of F in waters in Arizona, known to cause mottled enamel, range from 1 to 18 parts per million. The lower concentrations (1 to 2 parts per million) usually produce the milder types of mottled enamel, characterized chiefly by their soft and chalky

white appearance. Concentrations from 2 to 8 parts per million produce the more serious pitted-and-corroded types. The highest concentrations of F cause severe mottling of deciduous teeth (9). Differences in types of mottled enamel within a family and among users of the same water-supply are probably best explained by variation in the amount of water, and therefore of F, consumed during the period when calcification of the teeth is normally proceeding at the most rapid rate. It can not be considered a difference in susceptibility as such.

CONCLUSIONS. Mottled enamel, a fluorine injury, cannot be prevented by dietary improvement. Sodium fluoride, if present in food or water supply, exerts its maximum effect regardless of adequacy of diet. Attempts to nullify this effect in lower animals, or to lessen its severity, by supplying excesses of certain dietary essentials, have failed.

Suboptimal intake of any known dietary essential investigated did not result in increased susceptibility to the destructive action of flourine, although accompanying deficiency in dietary essentials necessary for normal tooth formation may result in teeth which are "doubly" defective.

The author expresses appreciation of the assistance of Miss Ruth Leverton and Miss Evelyn Blanchard in the conduct of these experiments.

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PERMEABILITY OF PLACENTA TO FLUORIDE

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In a recent article, Margaret C. Smith and H. V. Smith[1] reported the occurrence of a severe type of mottled enamel of all the deciduous teeth of a group of children in a certain locality. Prior to this discovery, the defect was commonly accepted as a lesion confined almost entirely to the permanent teeth. Very rarely has mottling of enamel been observed in deciduous teeth, and then only in molars, which are the last of the temporary set to calcify and erupt. Mottled enamel is now known to be a developmental lesion produced by the toxic action of fluorine (F), which exerts its effect during the period of calcification. This condition is endemic in regions in which the drinking water contains, by the Foster method of analysis, F in concentrations of one or more parts per million. Inasmuch as temporary teeth are believed to calcify chiefly before birth, whereas the permanent set undergoes calcification following birth, impermeability of the placenta to the F ion has been offered as an explanation of the relative immunity of the deciduous teeth to this defect. The discovery of mottled enamel in all deciduous teeth of children in a certain locality makes this explanation untenable. Analysis of the drinking water of the afflicted children revealed a F content of 12 to 18 parts per million, the highest concentration heretofore reported for any district. By the method of analysis employed, 1 part per million has been shown to be the toxic level for mottling of permanent teeth. It is a logical conclusion from this evidence that, in lower concentrations, F does not pass the placental barrier in amounts sufficient to affect the deciduous teeth, whereas in higher concentrations F is transferred across the placenta in harmful amounts.

In a more recent communication,[2] the same investigators presented

[1] M. C. Smith and H. V. Smith: *Science, 81,* 77, 1935.

[2] M. C. Smith and H. V. Smith: *J. Am. Den. Assoc., 22,* 814, 1935.

both clinical and experimental data leading them to question the permeability of the placenta even at the higher concentrations. They found that teeth of young rats, whose mothers received amounts of F in the food sufficient to mottle their own teeth severely, are normal at weaning time; but if the young are kept on a F-containing diet similar to that of the mother, the new incisor growth presents a mottled condition. Furthermore, they observed cases of mottled enamel of deciduous teeth in children whose mothers during pregnancy did not drink water high in F content. Such cases as these indicate that mottling of enamel in deciduous teeth is also a post-natal lesion; consequently, a considerable part of the calcification of enamel must go on after birth. They suggest that the rapidity of this calcification in temporary teeth as compared with that in the permanent set may explain why it requires a higher concentration of F to cause mottled enamel in the deciduous dentition. In view of the current interest in the problem of mottled enamel of deciduous teeth and the factors involved in its production, we report here the results of preliminary experiments on the permeability of dog placenta to the F ion. Although the number of pregnant dogs secured for experimental purposes was not large, the positive nature of the results to date is considered significant, as they demonstrate that at certain concentrations F is transferred from mother through placenta to offspring.

In our experiments, Na F in varying amounts was administered in the drinking water during the period of gestation. Tissues were obtained from fetuses or newborn, and mothers, and analyzed for content of F. The dried tissues were ignited carefully to avoid loss of F, which is appreciably volatile at bright redness. Standard methods of analysis were used. In the glass-etch test for qualitative presence of F, only definite etching when viewed in reflected light was taken as positive evidence. The quantitative estimation was made by the method of the Association of Official Agricultural Chemists.[3] The weighed sample was distilled at 135°C. with ground quartz and perchloric acid (60 percent), and the distillate titrated with standard thorium nitrate, using sodium alizarine sulfonate as indicator. Sections of the teeth of a pup from Bitch I, and of a fetus from Bitch III, were examined for evidence of abnormality due to F. Through

[3] D. S. Reynolds: *J. Assoc. Off. Agr. Chemists, 17*, 323, 1934.

an oversight sections of the pup from Bitch II were not made. *Table 1* gives the results of the chemical analyses.

Bitch I, given 5 p.p.m. (parts per million) in drinking water for 8 weeks, showed 0.18 percent of Na F in the analyzed specimen; no F in the fetus at term. In *Bitch II*, given 25 p.p.m. of Na F throughout gestation, there was a F content of 0.298 percent in her tissues. In her young at birth there was a concentration of 0.073 percent of F, indicating that the F ion passed through the placenta. In comparing the results obtained from *Bitches I* and *II* and their fetuses, it appears probable that absence of the F ion in the fetus of the bitch on the

TABLE 1

Occurrence of fluorine in experimental animals

ANIMAL	DOSAGE OF NaF	DURATION OF FEED	PART ANALYZED	GLASS-ETCH TEST	QUANTITY PRESENT
					percent
Bitch I	5 p.p.m.	8 weeks	Foreleg	Positive	0.180
Term fetus from Bitch I		Cancelled at birth	Whole fetus	Negative	None
Bitch II	25 p.p.m.	Duration of pregnancy	Foreleg	Positive	0.266
Term fetus from Bitch II		Cancelled at birth	Whole fetus	Positive	0.073
Bitch III	Abortive dose (50 mg.)	7.5 weeks	Foreleg	Positive	0.189
Aborted fetus of Bitch III		Aborted	Whole fetus	Positive	0.325

smaller dosage was due to fixation of all Na F in the maternal tissues rather than to any impermeability of the placenta. When the capacity for this fixation was exceeded, the F ion passed through the placenta and appeared in the fetus, as shown in *Bitch II* and her offspring. The data obtained from *Bitch III* and her aborted fetus strengthen this conclusion. In this case the mother received a relatively large dose over a shorter period (well along in pregnancy; abortion produced at least two weeks before term). The analyses showed a much higher percentage in the fetal than in the maternal tissues. This difference in concentrations of F, with the higher quantity in the fetus, may be an error, due to inequality of sampling—the moth-

er's sample was mostly bone; the entire fetus was used. It might be assumed, however, that more F is fixed in the fetus, owing to the rapidity of bone formation as contrasted with that of the mother.

The dosage of Na F for these dogs was chosen arbitrarily. In the case of Bitch II, on which this experiment had been run prior to the publication of the articles already mentioned, it is interesting to note that the analytic results for the fetus compare favorably with the occurrence of mottled deciduous teeth of children in a locality where the drinking water contains from 12 to 18 p.p.m. of F.

The results may be summarized as follows:

1. Sodium fluoride, administered in concentrations of 5 p.p.m. to dogs, during the last eight weeks of gestation, did not reach the fetus in concentrations sufficient to be detected by the analytical method used. Sections of the fetal teeth showed no marked differences from normal.

2. Sodium fluoride, administered in concentrations of 25 p.p.m. throughout gestation, appeared in the fetus in detectable and measurable quantities. No sections were made of the fetal teeth in this case.

3. Sodium fluoride, administered in large doses of increasing strength until abortion occurred, gave greater fluorine content in the fetal than maternal tissues. Sections through the molar teeth of the aborted fetus showed marked interference in enamel formation, as evidenced by almost total lack of enamel in the presence of a relatively large dentin formation, the clinical determinations being thus supported by the histological findings.

Further studies are in progress, to determine the threshold above which the fetus is affected by sodium fluoride given to the mother; also the possibility of fluoride transmission through mammary glands.

INTERNATIONAL ASSOCIATION FOR DENTAL RESEARCH

Proceedings of the Fourteenth General Meeting[1] Brown Hotel, Louisville, Kentucky

March 14 and 15, 1936

Compiled by WILLIAM J. GIES, F.A.C.D, *Secretary*[2]

Columbia University, New York City

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[1] The sessions of the Research Association preceded, by agreement, those of the Thirteenth General Meeting of the American Association of Dental Schools, in the same hotel, March 16–18. The members of each body were welcome at, and participated in, the sessions of both associations. The chairmen were the President, Dr. Theodore B. Beust, University of Louisville; the Vice-president, Dr. Paul C. Kitchin, Ohio State University; and the President-elect, Dr. William G. Skillen, Northwestern University. The annual informal dinner, served to members and guests on the evening of March 15 preceding the concluding session, was attended by 124 persons. [The President's address, abstracts of papers, and data of executive proceedings, etc., *at the general meeting in 1935*, were published in the *J. Den. Res.*, 1935–36, *15*, 139–232; June-Aug.]

[2] Most of the abstracts have been prepared by those who presented the corresponding papers. All editorial revisions have been verified by the authors.

I. Address of Retiring President[3]

Theodore B. Beust, D.D.S., M.D., F.A.C.D., School of Dentistry, University of Louisville, Louisville, Ky.

Life is a stern master. Probably one-third of civilized man's existence is devoted to the task of providing sustenance. "Enviable is he whose pursuit is fairly endurable. Lucky is he whose work is fairly congenial. Blessed is he who anticipates his daily duties with a pleasurable thrill." To the last category belong the few who are privileged to devote the major part of their time to research.

The investigator's immutable devotion to the solution of apparently insignificant problems presents an interesting study in human behavior. The urge to delve into the unknown implies the existence of a heritage extending back to the roots of the human race. Primitive man—even the human precursor—looked askance at the pranks of nature. Rain, lightning, thunder, and fire, according to the oldest records, were attributed to the benevolence or wrath of the supernatural. This signified the presence of a spirit of inquiry concerned with the relation between phenomenon and cause. This faculty of correlation must have been the factor that made the human race the intellectual superior of all creatures. The evolution of this spirit increased the mastery of man over the animate and inanimate. The passing of countless centuries saw the gradual creation of a soil embellished with mysterious alchemic laboratories and with centers of learning. Hippocrates, Euclid, Plato, appeared. The research man passed from the subconscious observer to the student of modern times. Empiricism, long a valuable adjunct of science, yielded to the methodical use of known laws or theories based on logic. *Accidental* discovery of truth, however valuable, no longer constitutes research. The concept now includes directed and sustained efforts for the solution of problems. No longer is it satisfying to know that a crowbar will help to move a weight. It is now asked that the principles be explained, and the result of forces applied to its handle be computed. Individuals following this line of work are dominated by mysterious inner forces. They find themselves in a state of submission bordering on slavery. Their workshop, whether a Rockefeller

[3] This, and the two addresses on pages 299–301, were delivered after the annual dinner at the sixth session (March 15), before the concluding executive proceedings (page 377).

creation or a corner in an attic, is a sanctuary pervaded with the great spirit of research. They know but one desire: the solution of their problem. Regardless of hurdles, unaffected by back-sets, unconscious of discomforts, and oblivious to the passage of time, the devotee, with unflagging interest, toils toward his goal. Archimides, during the siege of Syracuse, was killed by a soldier while he himself was so absorbed in mathematical calculations that his only remark was: "Do not disturb my diagrams."

The history of research exhibits a significant periodicity in the production of world-approved investigators. In looking backward we find, for example, civilization set in wonderment by the researches of Harvey, Pasteur, Lister, Virchow, and others. These men deserved the popular acclaim that was theirs. But let us not forget the unsung legion of research men whose life work contributed to the principles which brought forth the illustrious few. Great men are rarely, if ever, spontaneous creations. They are expressions of the cumulative research of a host of scholars, assembled, correlated and enlarged upon at propitious moments. The rate and manner of growth of biologic science in its earliest days is reflected in its recent development. For example, Aristoteles, in the 4th century before Christ, built on the Hippocratic literature, in itself a product of over a hundred years of research. Aristoteles created an independent biologic science by releasing biology from the surfdom of medicine. His teachings persisted through the dark ages, dominated the work of Albertus Magnus in the 13th century, enriched the writings of Wotton in the 16th century, and guided the tendencies of Cuvier in the 19th century. Thus Aristoteles' direct influence extended through a period of over twenty centuries. The mathematician and architect Robert Hooke, in 1667, sectioned bits of cork and wood and found cells. During the next one hundred and seventy years nearly a score of botanists and histologists perpetuated their names on the roll of science through contributions to our knowledge of cells. But it was not until 1839 that cell-science had progressed sufficiently to beget the conceptions of cell reproduction published by the botanist, Matthias Schleiden. Subsequent studies undertaken with his friend, the physiologist, Theodor Schwann, revealed the astonishing fact of the cellular origin of all plant and animal tissues.

Such experiences are common in the annals of science. They illus-

trate that an evaluation of any piece of original research is impossible. Yet such attempts are sometimes made. An editorial on caries research, in the last volume of a prominent dental journal, although agreeing "that here and there a little seed has been sown," expresses the view that "never has such a vast amount of literature been written with so little concrete achievement." It adds that, unless the problem is solved soon, "we as a profession will stand convicted of a dereliction of duty." The rate of progress in biologic science, and the volumes written on unsolved or controversial problems in allied fields, indicate that this editorial was written in a moment of thoughtlessness, without regard to the fact that modern caries research made its advent with Leber and Rottenstein's work in 1867. Between that time and the early part of the present century the question of caries etiology, owing to almost universal acceptance of Miller's enlargement of Leber and Rottenstein's work, was given little attention. Recent voluminous caries-literature attests the realization that none of the theories thus far propounded has satisfactorily explained all phases of the complex problem. The numerous current publications constitute a favorable sign, denoting activity in many schools of reasoning. Theories are propounded, modified, and overthrown. These are steps in the evolution of science. The world, ignorant of the sacrifices involved, profits from beliefs that lived but could not endure.

Confirmed research men often lack in sophistry. This is especially true of men intensively engaged in biologic research. The biologist deals with phenomena of organized life, but his concentration on special problems, often involving all of his waking hours and frequently his hours of rest, precludes close familiarity with the capriciousness of the human race. Subtle machinations of mind based on worldly desires are foreign to his nature. He is satisfied if left undisturbed. That is why he so often is found in positions at insignificant salaries, with industrial corporations and other institutions where he manipulates the push buttons that stimulate progress. Research men are rarely politicians. Their work depends on punctiliousness, which is incompatible with political accommodations. This "shortcoming" can conceivably prove detrimental to an organization. Deep concentration on problems stands in an inverse ratio to practicality. Outward manifestations of a group of men are apt to be swayed by

the psychic state of its components. Fortunately most of our members exercise caution in routine transactions. Our rapidly growing society has thus far had the coöperation of officers who took an active part in its scientific proceedings. Unfortunately, however, a few members attend meetings for devious purposes. They rarely, if ever, are seen in the open sessions. Individuals, alien to the aims of the society, unacquainted with, or even unknown to the majority of its workers, might, under these conditions, be seated in administrative offices. An opportunity for sounding a note of warning is given a retiring president but once. Observance of our motto, "research and nothing but research," will tend to keep our organization clean. Precise specification of our interpretation of research, coupled with a more rigid application of our rules governing admission, would also help.

The letters "I. A. D. R." have been welded for all time by the immortal spirit of research. The most sanguine dreams of the founder and advisor of this Association, William J. Gies, must long have been surpassed. The potentialities of the members, when properly led, will forever prove a substantial bulwark to the waves of adversity. I relinquish my gavel with confidence in the future and bid my successor God speed.

II. Introduction of President-elect

Isaac Schour, B.S., D.D.S., M.S., Ph.D., Secretary of Chicago Section; College of Dentistry, University of Illinois, Chicago, Ill.

To introduce Dr. Skillen is a pleasant assignment. It gives me an opportunity to tell publicly what I have felt and known regarding a friend who has been generous with his help and constructive criticism. His personality is quiet, unassuming, modest. Very few, therefore, know that he is an excellent athlete and equestrian, and possesses artistic talents in painting. Born in Toronto, our President-elect came to Chicago at the age of thirteen, when his first research problem was to obtain employment. The successful solution of this problem was accomplished on his fourteenth birthday. He completed his high-school training at night, and obtained the dental degree in 1911

at Northwestern University. Upon graduation, Dr. Skillen was appointed a member of the faculty in the Department of Operative Dentistry. Apparently his interest in the fundamental sciences and research motivated him to leave the operative department, for in 1914, we find him in the Department of Histology of the same institution, working as a staff member without remuneration and developing a modification in staining technique.

It was fortunate intuition that led Dr. Skillen to make this change, for dental histology, as an organized branch of scientific dentistry, owes much to the impetus given to it by Northwestern University. The first lecture-and-laboratory course ever given in this subject was taught at this institution, in 1896, by Dr. Frederick B. Noyes. In 1914, Dr. Noyes became a teacher at the University of Illinois. Dr. Skillen was then appointed instructor under Dr. Newton G. Thomas, who succeeded Dr. Noyes as the head of the Department. Four years later, when Dr. Thomas followed Dr. Noyes, our President-elect was selected to head the Department. Dr. Skillen has continued and furthered the progressive tradition and standards of the Department of Dental Histology of Northwestern University for practically twenty years. It is, therefore, proper to pay tribute to him and his colleagues who have carried on research, not only in recent years but in the years preceding the organization of the International Association for Dental Research—when research was less fashionable, and called for a special fortitude of mind and courage of conviction to carry on in the face of indifference and apathy in a large proportion of the dental profession.

Dr. Skillen's investigations have reflected the research trends that have become increasingly manifest in recent years. Although he is primarily a histologist, his studies cover, in addition to purely descriptive microscopic analysis, physiologic, pathologic, clinical and experimental problems. He has often alternated periods of concentration on technique, be it staining or experimental, with periods devoted primarily to critical analysis and reflection. A fine balance between work and thought is an important factor in successful research. "Arm-chair science" alone is not research, nor is technique or mechanical skill. A happy combination of these, however, does constitute science. With each problem, regardless of what it may be, a cultural background is essential in the unfolding of new facts. Sometimes a

given historical background, a principle in another branch of science, or a happy idea, catalyzes one's work so that it progresses quickly and effectively where otherwise it would remain inert and static.

The somewhat younger men in the field of dental research are deeply indebted to Dr. Skillen and his co-workers, who have maintained high standards of research during the adolescent period of storm and stress in dental research, and have prepared the way for Gies' monumental contribution in placing dental research on an organized and permanent basis of usefulness and expansion. There are many members in the dental profession who ten or twenty years ago regarded research as the concern of some queer and impractical individuals, but who today have a genuine respect for it. I am informed on good authority that, at a national dental meeting in 1905, an audience of several hundred dentists had dwindled down to thirty members when the program was turned over to a leading authority who was to speak on "the relation of the structure of enamel to the preparation of cavity walls and margins." In spite of the fact that the subject was concerned with important clinical applications, the reference to fundamentals in dental histology was sufficient to cause a mass exodus. Today, as a result of the persistent work of the relatively small group of dental investigators, in which our President-elect has played an important rôle, we can appear before the dental profession with a subject title that holds no promise of immediate clinical application, and still command an interested and attentive audience. Today it is possible to give a report on even an experimental study dealing with the incisor of the rat, and still have an audience after the lantern slides have been shown in the dark. This remarkable transformation within a period of less than thirty years has been made possible by the thorough and persistent work of educators and investigators like our former presidents and Dr. William G. Skillen, whom I now have the honor to introduce as the next President of this Association.

III. Inaugural Address

William G. Skillen, D.D.S., Dental School, Northwestern University, Chicago, Ill.

Presumably, in making an address such as this, one is expected to take some stand on current problems; to erect a platform. However,

I have no real desire to do this, at least at this time, or even give you as much as a single plank. One hears rather too much of platforms these days—structures which to me seem to be on a par with new-year resolutions, and which do not even make good firewood. I, myself, am not particularly fond of after-dinner speeches and, to be consistent, should spare you. Someone, in outlining the proper length for a speech, has very aptly said that it ought to be like a debutante's dress—long enough to cover all essentials and short enough to sustain interest. I am very sure that I am not a good enough designer to meet all of your varied tastes in this respect. Furthermore, Elbert Hubbard has stated that "oratory is a matter of toothsomeness." He related the story of one of the greatest orators, who was thrown from his artistic hobbyhorse when his "store teeth," in an impassioned moment, shot over the footlights and fell with a sickening clatter into the orchestra. Wanting to retain what little prestige I may have as a speaker, I certainly do not wish to invite such a contretemps this evening. Therefore, on all counts, it would be well to terminate here and now.

However, it would seem that it has become the rule at a time like this that something must be said. In searching for a subject, I necessarily first reflected upon this organization, its purpose, its character, and its possible outcome; and, in so doing, came to realize perhaps more definitely than ever before just what factors have contributed most generously toward its success. Looking in retrospect over the years that have elapsed since the beginning of this group, it would appear, perhaps, that at least two more or less inter-related principles have been consistently maintained. Indeed, it would seem, that in the minds of many, these principles have assumed the proportions of ideals. *First of these is unity of purpose.* Each and everyone is, and has been, working toward a common goal; motivated by the same incentive; eager at all times to catch new thoughts—as it were, to "put salt on the tail" of a new idea, for the purpose of adding to our fund of knowledge of the working of the human mechanism, with the hope or expectation that ultimately some benefit may accrue. And in this working toward a common end, there seem to have been perfect satisfaction and happiness in doing what one could without particular consideration of cost or expectation of reward.

But unity of purpose, although a valuable asset in any group, probably would not in itself have been sufficient. *There must also be coöperation.* To be of genuine advantage, all work must be reciprocal. We help ourselves by helping others, and man alone will acomplish little or nothing. All of his thoughts and actions have a direct relationship with others and men succeed only as they work together. This too seems to have been a prevailing idea among this group. It had never appeared that anyone has been particularly desirous of personal emolument but, as some one has put it, has rather shied from all the "foolish claptrap of place and power." Personal gain has been incidental. Mutual gain has been paramount. This complete mutuality is exemplified, I think, by the fact that our discussions, although at times dangerously charged, have terminated in complete understanding with appreciation rather than in animosity. New opinions always have been welcomed; constructive criticism, invited. And I believe the past has clearly indicated that everyone is big enough to bear no grudges, harbor no jealousies, and accept no insults. Rather, as Emerson so nicely puts it: we have found in getting together that "we talk better, we have the nimblest fancy, a richer memory and our dumb devil has taken leave for a time." What is more, it would seem that out of even some of the most heated disagreements have developed real friendships. For instance, just as an example: when one receives, from an individual with whom one has disagreed more or less strongly at times, a letter addressed, "Dear Brother Bill," it would seem to mean something—to me it appears to be about as friendly a gesture as one could wish. No doubt, most of you are able to recall such experiences for yourselves. Surely these are attributes of which any organization may be proud, and would seem fully to express Emerson's definition of friendship: that its "essence is one of entireness, total magnanimity and trust."

No one can deny that dental research has finally come to occupy a not-too-small place in the world of science. It is my feeling that this has been made possible and hastened by the complete unanimity of purpose, thought, and activity which has guided the members of this organization, together with their tolerant and friendly reciprocity toward each other. And I sincerely wish to see this attitude maintained. Therefore, it is my hope, even my firm belief, that this little

plank—which, in spite of myself, perhaps I have given you—will prove to be not a springboard, but a sufficiently strong foundation to assure even greater success than you have heretofore realized. I believe that this success will be furthered if our means of publication is preserved to us [*Journal of Dental Research*]. In conclusion, I believe we all owe a great deal to our much respected and far-sighted pilot, Doctor Gies, under whose guidance these things have been made possible.

IV. Index of Names of Participants, and Sequence Numerals of Corresponding Abstracts

Appleton, 61, 99; Armstrong, 15, 16, 40; Arnold, 8; Bale, 26; Bassett, 26; Bessey, 51; Beust, 3, 17, 72; Bibby, 94; Blackberg, 50; Blayney, 44; Bocak, 75; Boyle, 51; Brawley, 95, 96, 116; Breitner, 76; Brekhus, 15, 16, 40; Brown (J), 33; Bunting, 59; Cavett, 16; Chase, 11, 12; Cook, 58; Crowley, 4; Dahlberg, 6, 14; Davenport, 38; Dean, 97; Denton, 25; Dixon, 55; Doan, 63; Driak, 77; Edwards, 65; Eisenberg, 98; Fink, 5, 42; Finn, 26; Fitzgerald, 36; Forbes, 60; Freeman, 46; Gachet, 26; Gottlieb, 78; Grohs, 79; Grossman, 99, 100, 101; Gunkel, 67; Hall, 29; Hammond, 5, 42; Harrison, 30; Higley, 102; Hill, 8; Hine, 1, 2; Hodge, 7, 22, 26, 32; Hofer, 80; Hollander, 103; Homberger, 27; *Jeserich, 104; Kaplan, 107; Karshan, 105; *Kellner, 81; Kent, 64; Kesel, 44; Kitchin, 63; Klotz, 33; Kotanyi, 82; Krasnow, 106, 107, 108; Kronfeld, 41; LeFevre, 26, 32; Logan, 54; Losch, 38, 39; Lose, 26; Lowery, 53; Manly, 7; Mathis, 83; McBride, 9, 10, 30; McCoord, 26; McCrea, 20, 109; McDowell, 69; Messner, 71, 110; Miner, 68; Moore, 49; Morse (F), 38, 39; Myers, 34; Neuwirth, 111; Noyes (H), 45; Oartel, 48; Oblatt, 106, 107; Orban, 84; O'Rourke, 66; Paffenbarger, 37; Pichler, 85; Preissecker, 86; Price, 112, 113, 114; Rickert, 4, 55; Rittershofer, 13; Robb, 26; Robinson (H), 20, 26, 29, 56, 57; Rosen, 108; *Ryder, 115; *Schönbauer, 87; Schoonover, 37, 52; Schour, 47, 73; Schwarz, 88; Sedwick, 96, 116; Sicher, 89; Skillen, 74; Skinner, 35; Slanetz, 50; Souder, 52; Stafne, 31; Stein (G), 90; Stein (M), 62, 117; Sulser, 46; Swanson (E), 118; Swanson (W), 23, 24; Trauner, 91; Trevor, 18, 19; Tunnicliff, 5; Ulicny, 43; Van Huysen, 21, 26, 29; Van Kirk, 23, 24; Vinsant, 70; Wach, 44; Waugh (D), 28; Waugh (L), 28; Weinmann, 92; Werle, 11, 12; White, 59; Wills, 60; Wolbach, 51; Wolf (E), 9, 10, 30; Wolf (H), 93; Wolfsohn, 119; Youngburg, 120; Ziskin, 50.

V. First Session: Morning, March 14; Abstracts 1–12

1. Bimonthly observations of oral flora. *Maynard K. Hine, D.D.S., M.S., Rockefeller Fellow in Dentistry, School of Medicine and Dentistry, University of Rochester, Rochester, N. Y.* Method to determine variations in mixed flora, which reveals all morphological groups of bacteria present and their numerical relationships (Bibby: *J. Den. Res.*, 1935–36, *15*, 170), was adapted to show variations in bacterial

* No manuscript received for publication; read by title.

flora of two oral areas in ten healthy adults. Smears from chosen areas (usually buccal surfaces of upper right first molars, and distal surfaces of lower right third molars) made every two weeks for ten weeks. Bacteria were classed as *Fusiformis* (*nucleatus, polymorphus, dentium*), *Leptotrichia*, gram-positive and gram-negative cocci, gram-positive and gram-negative bacilli, and vibrios. Marked variations, from patient to patient, could not be correlated with any recognized differences in oral conditions. Variations also noted for same areas from same mouth at different times; in three cases changes developed so definitely that percentage of one or more morphological groups increased or decreased progressively for from six to ten weeks. By chance, one area chosen for study—caries-free at first—developed tiny cavity during period of observation. First percentage count showed cocci, 81; filaments, 15; bacilli, 3. Successive observations revealed steady change: in 10 weeks percentage counts were cocci, 60; filaments, 25; bacilli, 15. Occasionally flora of two different areas in same mouth showed simultaneous percentage changes in one or more morphological groups, but usually variations for each area were independent.

2. DAILY OBSERVATIONS OF BACTERIAL INHIBITION BY SALIVA. *Maynard K. Hine, D.D.S., M.S., Rockefeller Fellow in Dentistry, School of Medicine and Dentistry, University of Rochester, Rochester, N. Y.* Method of demonstrating inhibition of bacterial growth by saliva, using saliva-filled wells cut in seeded plates (Fleming, *Lancet*, 1924, *1*, 1303), carefully studied. Bacteria and media used were *M. lysodeikticus* (plain agar); *L. acidophilus* (galactose-whey agar); *Streptococcus*, beta-hemolytic type (Savita + 1 percent horse-serum agar); *Staphylococcus albus*, two strains; *Staphylococcus aureus* (Kulp's tomato-juice agar). Inhibition of bacteria, listed above according to decreasing susceptibility, varied so uniformly that it was concluded first two species indicated any variations in degree for different samples of saliva. Resting saliva from four patients, tested (27 of 38 days) against six bacteria listed, showed inhibitory powers that varied from patient to patient, and from day to day. Variations appeared to be haphazard, or dependent on undetermined factors. Because of difficulty in controlling all technical procedures that influenced width of zone of inhibition around saliva-filled wells, it was concluded that,

although method is limited in value, duplicate results to within ± 1 mm. may be obtained, if exact technique is used. Inhibitory "principles" were present in 91 percent of 1088 tests.

3. CONTACT-POINT CARIES. *Theodore B. Beust, D.D.S., M.D., F.A.C.D., School of Dentistry, University of Louisville, Louisville, Ky.* Common belief that enamel caries starts beneath plaque, or colony of acid-forming organisms, was controverted by results for 287 extracted teeth having caries on approximal surfaces. Of these, 246 displayed facets of interproximal wear. Of this number, 15 had caries both inside and outside of facet. Remainder exhibited caries within facet only. Practically all interproximal enamel-caries starts at contact point, in environment favoring generation of toxic ferments of a protein nature. Presence of acid-yielding plaques at contact point mechanically impossible. (To be published in *J. Den Res.*)

4. INFLUENCE OF EFFECTIVE MOUTH-WASHES ON BACTERIAL CONTENT OF MOUTH, AS ESTIMATED BY ATOMIZER METHOD. *Mary C Crowley, M.S., and U. G. Rickert, A.M., D.D.S., F.A.C.D., Dental School, University of Michigan, Ann Arbor, Mich.* Four popular mouth-washes tested on five individuals. Control counts made. Method previously reported (*J. Bact.*, 1935, *30*, 4). Results: appreciable decrease of oral bacteria (in six hours) after use of one mouth wash in dilution tested; two mouth washes decreased count for short time (three hours); one had no noticeable effect.

5. SIGNIFICANCE OF FUSIFORM BACILLI AND SPIRILLA IN GINGIVAL TISSUES. *Ruth Tunnicliff, A.B., M.D., E. B. Fink, Ph.D., M.D., and Carolyn Hammond, M.A., John McCormick Institute for Infectious Diseases, and Foundation for Dental Research of the Chicago College of Dental Surgery, Chicago, Ill.* Although smear preparations from ulcero-membranous and pyorrheal gingivites contain large numbers of fusiform bacilli and spirilla, Levaditi-stained sections of these tissues show that, while a few fusiform bacilli occasionally invade pyorrheal tissue, they never are present in numbers equal to those in ulcer-membranous conditions. No such invasion of spirilla observed in pyorrheal tissues as that seen in ulcero-membranous infections (Vincent's). Definite immunity-reactions (phagocytic) with fusiform bacilli demonstrated in blood of patients having pyorrhea

and ulcero-membranous gingivitis, indicating this organism may be pathogenic.

6. TREATMENT OF GANGRENOUS TUSKS OF AFRICAN ELEPHANT. *A A. Dahlberg, B.S.D., D.D.S., University of Chicago, Chicago, Ill.* Four-year-old male African elephant having infections in tusks was treated with several medications in use for gangrenous human teeth. Right tusk had lost all pulp tissue four inches from end, and responded to formo-cresol treatment in ten days. No recurrence of infection has been noted during succeeding thirteen months. Left tusk devoid of all pulp tissue, probe passing eighteen inches into canal. Nine treatments with formo-cresol, over period of four months, finally disinfected canal and periapical tissues. Reinfection occurred two months later. Dakin's solution, chloramine "T" (5 percent), formo-cresol, and camphorated phenol, although unsuccessful, have kept purulent discharge at minimum. Secondary dentin will probably fill and obliterate canal in right tusk as it does in normal ones. By continuance of drainage from left canal, and application of volatile antiseptics, it is hoped to keep process under control and prevent extension.

7. CENTRIFUGAL METHOD FOR QUANTITATIVE SEPARATION OF ENAMEL AND DENTIN. *Harold C. Hodge, B.S., M.S., Ph.D., Senior Rockefeller Fellow in Dentistry, and Richard S. Manly, B.S., M.A., School of Medicine and Dentistry, University of Rochester, Rochester, N. Y.* Centrifugal method devised for routine separation and purification of enamel and dentin in single teeth, making use of (*a*) liquid having density of 2.7, and (*b*) new type of separation tube having *outer* ordinary 15-cc. centrifuge-tube, and *inner* tube—prepared from test-tube by drawing out bottom and cutting to leave 3-mm. hole, top being flanged to fit rubber stopper, which rests on edge of centrifuge tube, to support end of inner tube about 1 cm. above bottom of centrifuge tube. After assembly is half filled with heavy liquid, the tooth, previously powdered in diamond mortar to pass 60-mesh screen, is transferred to liquid in inner tube, upper portion stirred gently, and whole centrifuged repeatedly. Dentin remains in inner tube, which may be lifted like pipette by closing top with thumb and dentin transferred to another centrifuge tube. Washing three times with CCl_4 and drying completes process. One separation requires

about an hour, entails total loss of about 3 percent of material, and gives enamel and dentin fractions each having purity of 99 percent, as determined by refractive-index method devised by authors. Purity of 99.9 percent in enamel product may be obtained by second separation with pure bromoform, with about 5 percent further loss of enamel. Dentin, 99.7 percent pure, may be obtained by another fractionation with liquid having density of 2.4 grams per cc. Cementum may be removed from dentin with liquid having density of 2.07 grams per cc.

8. SILVER NITRATE IN PREVENTION OF DENTAL CARIES. I. EFFECT UPON DECALCIFICATION OF ENAMEL. *Thomas J. Hill, D.D.S., F.A.C.D., Institute of Pathology and School of Dentistry, Western Reserve University, Cleveland, Ohio, and Francis A. Arnold, B.S., D.D.S., United States Public Health Service, Washington, D. C.* To study relative solubility of enamel treated with $AgNO_3$, used powdered enamel separated from dentin by method described by Armstrong and Brekhus. Material, after treatment with $AgNO_3$, subjected to action of weak organic acid, and rates of dissolution of treated and untreated enamels compared. Under these conditions, treated enamel becomes more resistant to action of weak organic acids. Organic content of enamel can combine with only small fraction of Ag that reacts with enamel. Reduction in solubility due to combination of Ag with more readily soluble inorganic matter in enamel.

9. BONE AND TOOTH FORMATION: GOMORI'S SILVER NITRATE METHOD. *E. Alfred Wolf, Ph.D., and T. F. McBride, D.D.S., Department of Biology and School of Dentistry, University of Pittsburgh, Pittsburgh, Pa.* With this method studied gradual calcification of bone, and formation of dentin and enamel in developing teeth, of whole embryos, and of skulls of new-born rats up to sixtieth hour. Insoluble lime salts were impregnated in fixed tissue before decalcification and sectioning; black deposits of Ag remained at site of Ca salts, thus giving clear and selective pictures of loci of calcification in thin microscopic sections. In long bones, deposition of perichondrial bone and ossification of intercellular substance strikingly shown. First traces of beginnings of calcification seen where three or more cells touched. In vertebrae, calcification begins at periphery of cartilage, in hyaline layers of fibroblastic origin; but is soon followed by Ca deposits in intercellular spaces of cartilage. In skull, dermal and cartilagenous

bone could be easily identified. Various elements of developing tooth-germ demonstrated in new and advantageous manner; Ca-developing structures easily discernible.

10. DEPOSITION OF CALCIUM IN DEVELOPING TEETH OF RATS. *T. F. McBride, D.D.S., and E. Alfred Wolf, Ph.D., School of Dentistry and Department of Biology, University of Pittsburgh, Pittsburgh, Pa.* Gomori's $AgNO_3$-method used to study Ca deposition in developing molars and incisors of embryo and new-born rats. In dentin formation, first deposition of Ca salts occurs on side of pre-dentin that borders on ameloblasts. No traces of Ag in odontoblasts, these cells being perfectly clear. In enamel formation, distal part of each ameloblast (containing nucleus) free from Ag deposits; in proximal part there was gradual accumulation of Ag—very dense toward end of cells, there forming apparently continuous black line running across ameloblastic layer. Under high magnification, "line" resolved itself into individual dots, one for each cell; did not connect ameloblasts with each other; was not thickened ends of intercellular substance; and did not close intercellular space—phenomenon intracellular. Upper part of enamel itself composed of layers parallel to surface that seemed to be derived from fusion of said intracellular dots. Formation of enamel thus appeared to be rhythmical intracellular process. Black deposits of Ag that replaced Ca salts during treatment did not mask structure of dentin and enamel under high-power (oil) magnification.

11. TESTS OF "LIGHT-AND-DARKNESS" HYPOTHESIS OF GENESIS OF TRANSVERSE PRISM-STRIPES. *Samuel W. Chase, A.M., Ph.D., and Jacob M. Werle, A.B., School of Medicine, Western Reserve University, Cleveland, Ohio.* Three groups of four white rats each kept under experimental conditions twelve weeks. *Group 1* exposed to direct day and night conditions of light in an open window. *Group 2* screened from actinic rays by panes of thick window-glass. *Group 3* kept in total darkness. Microscopical examination of ground longitudinal sections of all incisors showed no significant difference in incidence, appearance, size, or spacing of transverse prism-stripes. This regarded as conclusive evidence against theories that transverse prism-stripes are produced during presence (Asper) or absence (Gysi) of ultraviolet radiation.

12. TEST OF DIETARY RHYTHM AS POSSIBLE FACTOR IN GENESIS OF

TRANSVERSE PRISM-STRIPES. *Samuel W. Chase, A.M., Ph.D., and Jacob M. Werle, A.B., School of Medicine, Western Reserve University, Cleveland, Ohio.* Since about six transverse prism-stripes are formed daily in incisors of white rats, and since Richter has shown these animals eat at intervals of three to four hours, possibility of causative relation between ingestion of food and formation of cross-stripes tested experimentally. Normal dietary rhythm of *four* rats altered by supplying food for three-hour period, daily, for nine weeks. For *two* rats, food available for forty-eight successive hours followed by absence of food for twenty-four hours, out of each three days for month. For *two rats* (*controls*), food available constantly for nine weeks. Microscopical examination of ground longitudinal sections of all incisors showed no significant difference in incidence, appearance, size, or rhythm of deposition of transverse prism-stripes. Hence dietary rhythm alone must be excluded from consideration as causative factor.

VI. Second Session: Afternoon, March 14; Abstracts 13–24

13. DIMENSIONAL CHANGES DURING GROWTH AND DEVELOPMENT OF FACE. *Leslie F. Rittershofer, D.D.S., M.S., Dental School, University of Michigan, Ann Arbor, Mich.* Most striking feature of skull, at birth, is insignificance of face. Anthropometric measurements involving height, width, and depth, on collection of skeletal material of various ages in Museum of Anthropology (Univ. of Mich.), indicate direction, amount, and time of changes from early childhood throughout life. After birth, facial portion of head obviously grows much more rapidly than cranial. Measurements indicate growth-rate rises rapidly, though not uniformly, during childhood; tends to diminish to relative stability during maturity; finally, during senility, becomes atrophic or negative. Growth in facial height and depth greater than in width. Width change occurs mainly at posterior part. Growth faster backward than forward. Great posterior change (face and jaws) results from addition of new material. Thus, in lower jaw, greatest increase in dimension takes place from second molar region backward. Although greatest degrees of increase and decrease occur in bone that supports teeth, facial bones not involved in this function show identical trend, even in senility. Absorption of alveo-

lar bone not due entirely to loss of teeth. Age a factor, for which there is little compensation, if any. Briefly: face never static, dental portion least of all. Knowledge of time and direction of changes aids prognosis in restorative as well as corrective fields.

14. INHERITED CONGENITAL ABSENCE OF SIX INCISORS, DECIDUOUS AND PERMANENT. *A. A. Dahlberg, B.S.D., D.D.S., University of Chicago, Chicago, Ill.* In 18 of 46 members of one family, through four generations, all deciduous and permanent maxillary lateral incisors, and mandibular central and lateral incisors, missing congenitally. Radiographic studies of three having anomaly discussed. Wassermann and Kahn tests, and physical examinations, negative. In one child mandibular second bicuspid also missing; deciduous second molar present. Transmission of anomaly has been through both male and female when affected, but not through individuals having normal dentitions. Affected females produced both normal and affected males and females. Up to this time only one affected male has had children: of his 7, there were 1 normal male, 5 affected females, 1 normal female. Assuming sex-linkage, it would have to be on "X" chromosome; and anomaly would appear when one chromosome affected. This pattern would be applicable to present case—excepting unaffected daughter of affected male. There are possible explanations for this, but future offspring will have to be studied before definite statement can be made. If this fails to carry, anomaly in this particular case will be recognized as at least example of simple Mendelian dominant. (To be published in *J. Den. Res.*)

15. COMPOSITION OF MINERAL PHASES OF ENAMEL AND DENTIN. *W. D. Armstrong, Ph.D., and P. J. Brekhus, B.A., D.D.S., F.A.C.D., Dental School and Laboratory of Physiological Chemistry, University of Minnesota, Minneapolis, Minn.* To test our contention that mineral phases of enamel and dentin differ in composition, protein was removed (from enamel and dentin prepared from pooled sound teeth) by boiling specimens in 3 percent KOH in ethylene glycol, according to procedure of Crowell, Hodge, and Line (*J. Den. Res.*, 1934, *14*, 251). Percentage composition of purified mineral material from *enamel*: Ca, 36.41; P, 17.39; Mg, 0.24; CO_2, 3.07; from *dentin*: Ca, 33.69; P, 15.78; Mg, 0.89; CO_2, 4.76. From numerous analyses of sound enamel and dentin, respective empirical formulae of mineral

phases of enamel and dentin have been calculated—$MCO_3[M_3(PO_4)_2]_{2.5}$ and $MCO_3[M_3(PO_4)_2]_{4.1}$, where "M" represents both Ca and Mg.

16. COMPOSITION OF PROTEIN IN DENTIN. *W. D. Armstrong, Ph.D., P. J. Brekhus, B. A., D.D.S., F.A.C.D., and J. W. Cavett, Ph.D., Dental School and Laboratory of Physiological Chemistry, University of Minnesota, Minneapolis, Minn.* Protein from dentin isolated by dissolving dentin in HCl and precipitating with trichloracetic acid. N content: 15.11 percent. Van Slyke N-distribution analysis yielded following illustrative percentage results: ammonia-N, 3.61–3.60; total basic-N, 33.17–32.60; total filtrate-N, 62.61–64.43; total recovered-N, 100.77–101.36.

17. MICROSCOPIC ANATOMY OF ENAMEL. *Theodore B. Beust, D.D.S., M.D., F.A.C.D., School of Dentistry, University of Louisville, Louisville, Ky.* Discussion of relation of inter-rod space-system (previously described by author) to striae of Retzius. With aid of plaster models showing incremental deposition of enamel, proof of fallacious interpretations of stratification and hypoplasia presented, adding convincing new anatomical evidence of identity of capillary-space system and striae of Retzius. (To be published in *J. Den. Res.*)

18. FORMATION OF LINES OF RETZIUS IN HUMAN ENAMEL. *Frederick A. Trevor, D.M.D., Dental School, Harvard University, Boston, Mass.* Investigation of decalcified stained sections, compared with ground sections of enamel, revealed rhythmic notch-formation in each enamel rod, resulting from antagonistic pressures from within and without tooth germ. (*a*) Resistance pressure from *without* tooth germ increases as growth pressure increases from *within* at regular intervals, causing definite *downward* path of ameloblast coincidental with calcification of portion of rod already formed. (*b*) Release of pressure from surrounding jaw-structures allows *upward* trend of ameloblast, until accumulation of resistance factors results in repetition of cycle. This downward and upward trend results in *deflexure notch* in enamel rod. Deflexure notch upon notch throughout enamel-cap produce *deflexure steps* and *deflexure furrows*. Between notch of one rod and next rod appears thickened area of inter-rod substance in semilunar form. This *matrix semilune*, being darker than rod, gives appearance of line or band when seen in aggregate, and is line

or band of Retzius. Rhythmic occurrence of notching appears to correspond to approximate monthly interval in formation of enamel. New terminology above: matrix semilune, enamel-rod deflexure-notch, deflexure steps, deflexure furrows.

19. FORMATION OF ENAMEL TUFTS. *Frederick A. Trevor, D.M.D., Dental School, Harvard University, Boston, Mass.* Embryonic decalcified stained sections, compared with ground sections of adult human teeth, showed these developments in enamel-tuft formation: (*a*) invagination and curvature in growth of enamel organ. (*b*) *Major* and *minor wrinkle-crest* formation at amelo-odontoblastic junction. (*c*) Tearing of inter-columnar bridges between ameloblasts. (*d*) *Expansion-crevice* formation at apex of major wrinkle-crest. (*e*) Entrance of cells of stratum intermedium as auxiliary or *secondary ameloblasts*. (*f*) Alignment of auxiliary ameloblasts along side of *primary ameloblasts*. (*g*) Formation of *wrinkle-crest line* by means of ameloblastic basement membrane, starting from major wrinkle-crest and developing along this line as enamel forms. (*h*) Extension of neighboring auxiliary ameloblasts along other primary-ameloblast sides in various directions. Some thickening of inter-rod areas due to discrepancies in space between auxiliary ameloblast top-plates and primary-ameloblast inter-columnar bridges. Wrinkle crests most common in cross sections of teeth, and at apices of sagittal sections where curves have shortest radius. Slight lateral-expansion growth-tension on inter-columnar bridges of ameloblasts, as they proceed, causes increase in width of peripheral portion of enamel rod. Expansion crevices, in which tufts are found, responsible for increase in enamel surface-area over smaller dento-enamel junction-area. New terminology above: wrinkle crests, (a) major, (b) minor; dento-enamel junction cone, (a) major, (b) minor; wrinkle-crest lines, (a) primary, (b) secondary; expansion crevice; ameloblasts, (a) primary, (b) secondary; enamel rod, (a) primary, (b) secondary.

20. DENTINAL PROJECTIONS IN MARSUPIAL ENAMEL. *M. W. McCrea, D.D.S., and Hamilton B. G. Robinson, D.D.S., Rockefeller Fellows in Dentistry, School of Medicine and Dentistry, University of Rochester, Rochester, N. Y.* Although demonstrations of dentinal tubules in marsupial teeth and comparisons with human teeth have been made by previous authors, very little fresh material has been

used; and ensuing possible occurrence and misinterpretation of artifacts not entirely eliminated. Studied teeth of six-year old *Macropus rufus*, in ground, decalcified, and cleared sections, some previously injected with basic fuchsin under negative pressure; others, stained with hematoxylin and eosin after grinding. Dentinal projections, traced between enamel rods, reached almost to external enamel surface. Bulbous endings morphologically comparable to enamel spindles in human teeth. Technique of clearing, permitting examination of thick slabs, shows scarcity of lamellar-like structures when compared with thin ground-sections, strongly suggesting that in many instances these are artifacts produced in grinding. Observation of dentinal projections, stained via pulp in thick sections, precludes misinterpretation of artifacts.

21. ROENTGEN-RAY ABSORPTION PER UNIT-THICKNESS OF DENTIN. *Grant Van Huysen, D.D.S., Rockefeller Fellow in Dentistry, School of Medicine and Dentistry, University of Rochester, Rochester, N. Y.* Using densitometric roentgen-ray absorption method described by Warren et al., dentin in 160 tooth slabs measured. Average absorption, per unit of thickness, of coronal dentin, 0.96 mm. Al.; for root dentin, 0.92 mm. Al. No difference shown in absorption for dentin in teeth from males and females, nor in carious and non-carious teeth. Age of dentin correlated with roentgen-ray absorption—as a rule, between 15 and 35 years, older dentin having higher average absorption than younger dentin. This increase amounted to about 4–5 percent for coronal dentin; 6–7 percent for root dentin. Decrease in magnitude of absorption-values occurs for dentin of individuals more than 35 years old—amount not determined.

22. MICROHARDNESS OF DENTIN. *Harold C. Hodge, B.S., M.S., Ph.D., Senior Rockefeller Fellow in Dentistry, School of Medicine and Dentistry, University of Rochester, Rochester, N. Y.* Rereading microcuts on 20 teeth after half-day, widths were reproduced to approximately 0.2 micron (average), representing range in microhardness of 5–8 units. Rereading microcuts on 25 teeth after 90 days, widths were all less (with one exception) by average of approximately 0.6 micron, corresponding to average microhardness 15–20 units greater. Lessened width tentatively attributed to removal, on standing, of traces of tissue on each side of microcut. Paired cuts in opposite

directions, in same area of tooth slab, gave almost as good correspondence of microhardness as would be found for rereading given microcut. For 19 teeth having transparent secondary coronal dentin, microhardnesses read from same microcuts showed that, on average, transparent dentin is about 25 units softer than normal-appearing dentin in same crown. This average difference probably without significance, since secondary dentin presumably varies from tooth to tooth, owing to systemic or other conditions at deposition and later.

23. RESULTS OF CULTURING 1800 PULPLESS TEETH. *W. F. Swanson, B.S., D.D.S., M.S., F.A.C.D., and L. E. Van Kirk, A.B., D.D.S., M.S., F.A.C.D., Dental School, University of Pittsburgh, Pittsburgh, Pa.* Bacteria, if present in pulpless tooth, would presumably be found in necrotic material in dentinal tubules and, if this were cultured in proper medium, organisms would be recovered. Used only extracted pulpless teeth, which were usually received on day of extraction; some, two or three days later. Root fillings of various kinds present in most teeth. No precautions used against contamination during extraction. Tooth immediately placed in gauze saturated with alcohol, rubbed thoroughly, and dropped into alcohol; flamed several times; and wrapped in sterile gauze. With sterile forceps, apex and mid-root portions cut off, and cultured in brain-heart infusion medium. In positive cases, pure cultures of diplo-streptococci (non-hemolytic) obtained. Positive cultures resulted for enough x-ray-negative teeth to warrant statement that absence of x-ray evidence does not guarantee sterility in pulpless teeth. General statistical data appended:

Culture	*Root-filled*		*Not root-filled*		*Total*	
	No.	*Percent*	*No.*	*Percent*	*No.*	*Percent*
Positive	1176	96.4	569	97.8	1745	96.8
Negative	44	3.6	13	2.2	57	3.2
Total	1220	100.0	582	100.0	1802	100.0

24. EXPERIMENTAL ENCEPHALITIS IN RABBITS: INTRAVENOUS INJECTION OF STREPTOCOCCI FROM PULPLESS TEETH IN HUMAN ENCEPHALITIS. *L. E. Van Kirk, A.B., D.D.S., M.S., F.A.C.D., and W. F. Swanson, B.S., D.D.S., M.S., F.A.C.D., Dental School, University of Pittsburgh, Pittsburgh, Pa.* While experimenting on effect of strep-

tococci from pulpless teeth when injected into rabbits, unusual localization occurred—patient: adult male, 77 years old; encephalitis. In effort to remove all possible foci of infection, several pulpless teeth extracted, cultures yielding gram-positive streptococci. Injected intravenously into rabbit, animal showed definite disturbance of central nervous system—loss of neuro-muscular control, twitching, paralysis, death. At post-mortem, culture of cerebro-spinal fluid yielded streptococci, which, injected into another animal, gave similar results. Repeated through six successive animals, streptococci retained affinity for central nervous system. In other cases, such as arthritis and colitis, streptococci never recovered from spinal fluid of experimental animals. Rosenow and others produced disturbances in central nervous system of rabbits by injecting streptococci directly into brain. In present study, organisms localized in central nervous system, causing profound disturbances in motor control, after indirect *intravenous* injection.

VII. Third Session: Evening, March 14; Abstracts 25–33

25. Research in dental history. *George B. Denton, Ph.D., Dental School, Northwestern University, Chicago, Ill.* Attention called to dental history as field for research. Technique of investigation briefly sketched, and exemplified by problems in history of dental porcelain. Most necessary sources for dental history available, but not thoroughly studied by competent investigators. Critical methods of establishing facts from documents not largely employed. Dental history mostly confined to attempts to determine qualitative facts, such as priority in discovery and invention. More important quantitative generalizations, regarding dissemination of knowledge and prevalence of practices, neglected.

26. Correlated clinical and structural study of hereditary opalescent dentin. *Harold C. Hodge, B.S., M.S., Ph.D., Senior Rockefeller Fellow in Dentistry; for a group including G. B. Lose, S. B. Finn, F. S. Gachet, S. H. Bassett, R. C. Robb, Grant Van Huysen, H. B. G. Robinson, M. L. LeFevre, W. F. Bale, A. B. McCoord, and others, School of Medicine and Dentistry, University of Rochester, Rochester, N. Y.* Hereditary nature of opalescent dentin illustrated and evidence offered that it may be quantitatively variable. Thor-

ough clinical work-up revealed no abnormality common to all patients, save opalescent teeth. Blood chemistry revealed essentially normal conditions, excepting plasma phosphatase in children—2 to 5 times normal. Other calcified structures normal-appearing in radiographs. Structurally, teeth have almost no pulp-chamber space, and contain dentin with vestigial dentinal tubules frequently running almost at right angles to normal-tubule course. Enamel frequently white and appears normal. Small roots and soft dentin predispose to fractures. Almost every mouth one or more apical abscesses. Microhardness of opalescent dentin very low—about 70 (normal dentin, 120); interpolated values per unit thickness (x-ray absorption) also very low—about 0.65 (normal dentin, 0.95). Chemically, opalescent dentin has abnormally high percentage water-content—about 15–20 (normal, 8–10); low inorganic content—about 70–75 (normal, 80–85); but essentially normal contents of Ca, P, and CO_2 (ash). In x-ray-diffraction studies, no difference noted in molecular constitution of inorganic parts of opalescent and normal dentin.

27. CALCIUM METABOLISM: VITAMIN-D FOODS COMPARED WITH COD-LIVER OIL. *A. W. Homberger, M.S., Ph.D., School of Dentistry, University of Louisville, Louisville, Ky.* In support of direct bearing of vitamin D on caries, numerous reports by outstanding workers cited. Recent endeavors to incorporate vitamin D into foods by various methods discussed. Influence, on rats, of milk fortified by irradiation, milk from cows fed irradiated yeast, and milk fortified with cod-liver-oil concentrate shown, and results for content of Ca in bones and blood presented. Findings indicated properly fortified milk excellent means of incorporating protective factor into daily diet.

28. DENTAL OBSERVATIONS AMONG ESKIMO. VI: BACTERIOLOGIC FINDINGS IN MOST PRIMITIVE AND POPULOUS NATIVE DISTRICT OF ALASKA. *L. M. Waugh, D.D.S., F.A.C.D., and Donald B. Waugh, Junior Student, School of Dental and Oral Surgery, Columbia University, New York City.* Study made of about 2000 Eskimo in Kuskokwim-Hooper Bay region of Alaska, which embraces some 5,000 natives existing in truly primitive state. A few had never before seen white person. Subsist on protein and fatty foods almost exclusively, only exceptions being few berries in some sections; in others, a little

vegetable in form of rush or reed butts. They have, as a rule, no carbohydrates, fruit or vegetables. Results completely corroborated previous findings in Labrador, North Bering Sea, and Arctic Alaska, concerning caries-free mouths and large well-shaped jaws with sturdy, strongly supported teeth in excellent occlusion. In about 10 percent of caries-free mouths, dental arches poorly developed, some teeth, especially incisors, being in mal-position; but in practically every such case, sequelae of other skeletal disturbances in childhood evident. Practically all primitive Eskimo mouths filthy. Study of lactobacilli made of 69 selected mouths. Bacterial incubator transported to settlement, and cultures and slides made in field. Attempts to follow Hadley's quantitative technic (plating measured quantities of paraffin-stimulated saliva on Kulp's tomato-agar) unsatisfactory. Qualitative results presented. Sixty-three of 69 mouths caries-free. Cultures obtained from six natives living within ten miles of Bethel, where four traders are well stocked with "civilization foods." Recent contamination of caries-free mouths might have occurred, because of lack of cleanliness with eating utensils in transient fishing camps where natives from many villages foregather. Of 54 cases having no caries, gram-positive rods found in 7; of these 7, teeth had been extracted in 2. Of 8 cases having caries, gram-positive rods found in 2; caries limited in all cases to two or three cavities per mouth. In other words, in 85 percent (plus) of *caries-free* cases, lactobacilli entirely absent; in 14 percent (plus) some lactobacilli present. Study will be continued next summer (1936) and until complete bacteriological, chemical, and nutritional surveys can be made. This is first recorded field work in bacteriology among Eskimo, and must be regarded as preliminary. Refinements growing out of actual experience in field are anticipated, and present report will, undoubtedly, be modified as result of further study. Slides amplifying text shown. (Previous reports in this series, presented to this Association, published in *J. Den. Res.*, 1928–33, *8–13.*)

29. ALVEOLAR ATROPHY IN DOGS. *J. Frank Hall, B.S., D.D.S., Hamilton B. G. Robinson, D.D.S., and Grant Van Huysen, D.D.S., Rockefeller Fellows in Dentistry, School of Medicine and Dentistry, Rochester, N. Y.* In large dog having very sore mouth (from animal house, this School), anterior teeth were broken and abraded, owing

to constant chewing on metal cage, and maxillary premolars were missing. Non-occlusion of incisors, mandibular premolars, and third molars; also much calculus about all teeth, and extensive atrophy of alveolus and supporting soft tissues. While secondary factors cannot be excluded, non-occlusion was apparently primary etiological factor in prevailing oral conditions. *Blood test*: 50 percent red cells, 140 percent hemoglobin. *Autopsy*: only slight thickening of mitral valve, petechial flecks on omentum, atrophy of spleen, minute abscesses on surface of kidneys. *Microscopic examination*: reduction of alveolar process, and many multinucleated osteoclasts—not proportionately greater in areas of pronounced resorption, and apparently not alone accounting for removal of bone tissue. Epithelial attachment: proliferated to apex of some teeth. Beneath crevice epithelium: small leucocytes increased in number. Compensatory hypercementosis noted. Sharpey's fibers, stained with hematoxylin-eosin, remarkably clear.

30. HEREDITARY OLIGODONTIA. *T. F. McBride, D.D.S., W. J. Harrison, A.B., and E. Alfred Wolf, Ph.D., School of Dentistry and Department of Biology, University of Pittsburgh, Pittsburgh, Pa.* Present study shows oligodontia, congenital absence of teeth, hereditary. Inheritance traced through four generations of a family of which eleven members showed abnormality. Four members of two youngest generations thoroughly examined, and casts and x-rays obtained. In maxillae, second incisors (except in one case), premolars, and third molars *missing*; *also*, in one member, right cuspid and right second molar. In mandibles, second premolars and third molars *missing*; *also*, in *one member*, right second incisor; in *another*, second incisors, first premolars, and second molars; and in *a third*, first premolars and second molars. Oligodontia in this family was fairly uniform; that is, teeth most commonly missing were second incisors, premolars, and third molars.

31. TUBERCULOMA INVOLVING UPPER MOLAR TOOTH. *Edward C. Stafne, D.D.S., Section on Dental Surgery, Mayo Clinic, Rochester, Minn.* Male patient, 63 years old, complained of pain in upper left first molar; had been filled several times. Roentgenogram revealed what appeared to be radicular cyst involving roots of tooth. Physical examination did not suggest any organic disturbance; patient had no

other complaints—no known contact with tuberculosis. When extraction was attempted, contents of bony cavity appeared to be firmly adherent to roots. Microscopic examination of contents: diagnosis—tuberculous, infected granulation-tissue. Sections stained for *Mycobacterium tuberculosis* revealed a few acid-fast bacilli. Microscopic examination of decalcified sections: lesion continuous with periodontal membrane of tooth. In view of pathologic findings, patient was returned to examining physician for further investigation. Subsequent roentgenographic examination of thorax revealed patient once had pleurisy at right base, and tuberculosis of right apex, of lung; no evidence of active tuberculosis. Lesion of jaw might have been related to former tuberculous condition in right apex of lung. Case raises question whether, in rare instances, tuberculous lesions may not be dental in origin. If it was dental in origin, most probable port of entry was through deep caries in upper left first molar. Such entry might have taken place at early age. (Published in *J. Am. Den. Assoc.*, 1936, *23*, 1694.)

32. ARE ANY CHANGES IN CHEMICAL COMPOSITION OF HUMAN TEETH DUE TO PHYSIOLOGICAL STIMULI? *Marian L. LeFevre, B.A., M.S., and Harold C. Hodge, B.S., M.S., Ph.D., Senior Rockefeller Fellow in Dentistry, School of Medicine and Dentistry, University of Rochester, Rochester, N. Y.* In lower jaw of patient who chewed exclusively on left side, teeth of that side contained less inorganic matter and Ca, had higher residue-solution number, were softer, and absorbed less roentgen radiation, than those of right side. Assuming abnormally high blood-flow in teeth of left side, findings are consistent with principle that calcification occurs best in tissues having low blood-supply. (To be published in *Den. Cosmos.*)

33. CHEMISTRY OF MIXED HUMAN SALIVA. II: SODIUM, POTASSIUM AND CALCIUM IN SALIVAS PRODUCED AT WIDELY VARYING RATES. *J. B. Brown, Ph.D., and N. J. Klotz, B.S., M.D., Medical School, Ohio State University, Columbus, Ohio.* Na, K, and Ca determined in three groups of mixed human salivas (six specimens in each group) produced at rates averaging 243, 143, and 61 cc. per hour, respectively. Na increased greatly, and Ca slightly, with rate of secretion. Values for K remained practically constant for all groups. Saliva produced at higher rates contains more NaCl, and less Na and K phosphates, than when secreted at lower rates.

VIII. Fourth Session: Morning, March 15; Abstracts 34–49

34. Hydrostatic-pressure method for determining surface tension of liquid metals. *Raymond E. Myers, D.D.S., B.S., School of Dentistry, University of Louisville, Louisville, Ky.* In experiments with mercury, cylindrical glass vessel, with small hole in center of bottom, was gradually filled until mercury began to flow through opening. Height to which mercury was raised, before flow started, determined. This value, together with radius of hole and density of mercury at observed temperature, permits determination of surface-tension. In experiments with high-fusing metals, special crucibles of Alundum, with circular holes in bases, were filled with different amounts of molten metal, and heights of columns measured after metal had solidified. Height to which metal could be raised, before flowing through opening, thus determined.

35. Contraction of investment mold during casting. *E. W. Skinner, Ph.D., Dental School, Northwestern University, Chicago, Ill.* Casting error, introduced by thermal contraction of inlay mold from time of removal from eliminating furnace to completion of casting, studied. Time-temperature cooling-curves of five commercial inlay-investments obtained, by investing wax pattern, containing thermo-junction, in usual manner, and heating to casting temperature of 1300°F.; after removing each mold from furnace, rate of cooling determined. Thermal contractions of same investments then determined with conventional fused-quartz thermal-expansion equipment. Two determinations correlated in terms of percentage contraction and time, results being shown in accompanying summary of data for contraction of casting mold with time:

Investment	*Casting time: contraction less than 0.01% Minutes*	*Casting time: contraction not greater than 0.05% Minutes*	*Casting time: contraction not greater than 0.10% Minutes*
A	0.75	1.9	2.5
B	1.25	2.2	3.0
C	1.50	2.6	3.5
D	0.60	1.2	1.7
E	1.50	2.5	3.7

36. Comparison of physical properties of high-, medium-, and low-fusing dental porcelains. *Percy A. Fitzgerald, D.D.S.,*

Dental School, Northwestern University, Chicago, Ill. Specific gravity, volume shrinkage, and modulus of rupture, determined for eight popular kinds of high-, medium-, and low-fusing dental porcelains, with results shown in *table 1.*

37. PHYSICAL PROPERTIES OF DENTAL SILICATE CEMENTS. *George C. Paffenbarger, D.D.S., F.A.C.D., Research Associate of the American Dental Association, and Irl C. Schoonover, Ph.D., Assistant Chemist, National Bureau of Standards, Washington, D. C.* In general, dental silicate cements are complex alumino-silicates containing Ca, Na, Mg, F, and P as major constituents. Of eleven cement powders, eight contained fluorine; beryllium present in two. Analysis of the liquids not completed. Method of preparing specimens for determinations of physical properties carefully controlled. By specifying consistency of mix of unset cement, temperature of mixing slab, time of mixing, and rate and method of incorporating powder into liquid, specimens giving reasonably uniform values produced. Consistency of mix adopted as standard represents that in actual dental practice. Amount of powder (grams per 0.4 cc.), required to produce standard consistency, varied from 0.65 to 1.50 grams, depending upon cement. Temperature variations caused pronounced effects on consistency measurements; variations in relative humidity, comparatively slight effects. Compressive strength increased with time; about one-third of maximum developed in 15 minutes; variations in powder-liquid ratio caused surprisingly small differences. Cements immersed in distilled water for six months slightly weaker than in natural saliva. Tests for determining time of setting, pH, solubility, dimensional change on setting, optical and other properties, in progress. It is believed sufficient data will be available to define relatively satisfactory silicate cement by particular physical and chemical tests embodied in specification. (To be published in *J. Amer. Den. Assoc.*)

38. ACIDURIC ORAL FLORA IN GROUP OF CHILDREN. *F. W. Morse, Jr., B.S., M.D., P.K. Losch, D.D.S., and S. E. Davenport, 3rd, A.B., Dental School, Harvard University, Boston, Mass.* In study from special children's group, at each visit of child duplicate cultures taken on pH 5.0 broth; smeared and also plated after 48-hour incubation. Bacteriological observations recorded during 342 child-years. For 98 children, there was correlation between caries and aciduric bacteria

in 52 percent of observed years; 19 percent of child-years showed complete reversal in this relationship, with no correlation; 29 percent gave indecisive results, both positive and negative cultures being ob-

TABLE 1

Physical properties of popular dental porcelains

GROUPS OF PORCELAIN	FIRING TIME	SP. GR.	VOLUME SHRINKAGE	TRANS. STRENGTH
			percent	
A(H.F.)	10 min. to 1900 5 min. to 2200 5 min. to 2400 5 min. soaking	2.32	32.2	7800
B(H.F.)	10 min. to 1900 5 min. to 2200 5 min. to 2375 5 min. soaking	2.33	31.7	7900
C(H.F.)	10 min. to 1900 5 min. to 2200 5 min. to 2400 5 min. soaking	2.32	32.2	8200
D(M.F.)	10 min. to 1800 5 min. to 1950 5 min. to 2100 5 min. soaking	2.30	32.6	7100
E(M.F.)	10 min. to 1800 5 min. to 2000 5 min. to 2200 5 min. soaking	2.32	34.6	8300
F(L.F.)	10 min. to 1600 5 min. to 1750 5 min. to 1850 5 min. soaking	2.29	39.5	3700
G(L.F.)	10 min. to 1600 5 min. to 1700 5 min. to 1800 5 min. soaking	2.32	33.3	7700
H(L.F.)	10 min. to 1600 5 min. soaking	2.50	37.4	7000

tained during year. The 7-year-old group of 52 children (age of highest incidence) showed 87 percent of caries-free negative for aciduric bacilli in all observations. In group having 6 or less cavities: 33 percent positive; 67 percent either negative or indecisive. In

group having 6 to 11 cavities, from birth to age 7, 65 percent showed positive cultures. In group with highest incidence, 100 percent gave positive cultures.

39. INCIDENCE OF CARIES AND RELATION TO PRENATAL AND EARLY PEDIATRIC CARE. *Paul K. Losch, D.D.S., and Fred W. Morse, Jr., B.S., M.D., Dental School, Harvard University, Boston, Mass.* Incidence of caries in selected group of Boston children of pre-school and early-school age is lower than in any group of American children previously reported, where no specific measures of prevention had been taken. In relation to prenatal care, 94 percent of mothers whose children are caries-free had medical supervision during full term of pregnancy, while only 63 percent of mothers of highly susceptible children had such care. Regular medical supervision of care and feeding during infancy received by 88 percent of caries-free class as compared to 38 percent of highly-carious group. Caries in children apparently influenced by illness during pregnancy and infancy. No single factor determines caries immunity, but appears to be influenced by measures commonly accepted as good hygiene during pregnancy and infancy.

40. GNATHODYNAMOMETER. *P. J. Brekhus, B.A., D.D.S., F.A.C.D., and W. D. Armstrong, Ph.D., Dental School, University of Minnesota, Minneapolis, Minn.* History and principles of previous instruments for measuring force developed in mastication reviewed. New instrument, based on hydraulic principles, constructed. Force applied between entire complement of teeth, or between opposing pairs of teeth, transmitted to compressible chamber (Sylphon) and in turn, through liquid column, to pressure gage.

41. DISTRIBUTION OF BONE MARROW IN HUMAN JAWS. *Rudolf Kronfeld, B.S., D.D.S., M.D., Foundation for Dental Research of the Chicago College of Dental Surgery, Chicago, Ill.* Distribution and possible significance of various types of bone marrow studied in sections through human jaws. In most adult jaws, fat marrow is main type, especially in edentulous areas. Islands of red marrow frequently encountered, as reported by Box; in some instances this cellular marrow appears to be reaction to patient's terminal illness. Fibrous marrow, confined to vicinity of dental-apical infections, seems to be specific response of jaw bone to chronic inflammation of long standing.

42. ATTEMPTS TO CULTIVATE SPIRILLA FROM CASES OF PLAUT-VINCENT INFECTION. *E. B. Fink, Ph.D., M.D., and Carolyn Hammond, A.M., Foundation for Dental Research of the Chicago College of Dental Surgery, Chicago, Ill.* Clinical material consisted of four cases of Plaut-Vincent infection; one of osteomyelitis of jaw, in which smears showed almost pure growth of coarse spirochetes; one of gangrene of face, complicating severe sinus infection in child; one of chronic infection of submaxillary duct, complicating stone in gland; and one of severe diabetes with stomatitis. Patients brought from clinic to laboratory, and material inoculated directly into various media. In some cases scrapings made with bacteriologic needle; in others sterile cotton-swabs used; in one case of Vincent infection, mouth rinsed several times with sterile saline solution, washings passed through Berkefeld filter (No. N), and filtrate inoculated into various media. Anerobic conditions of growth obtained by Wright pyrogallic-sodium-hydroxide method. *Media used*: Noguchi's serum-water in tall wide-tubes, plus small piece of fresh, sterile, rabbit kidney just before inoculation; various combinations of ascitic fluid with veal-infusion agar; rabbit serum, with and without addition of tissue; gentian violet (1–10,000) added to plain agar and sheep-serum mixtures; phosphate agar, with addition of ascitic fluid; malt-dextrose agar slants; anerobic blood-agar plates. With Noguchi technique, spirochetes survived five weeks, then disappeared. Impossible to get rid of streptococci—outgrow all other organisms. Spirochetes disappear after third subculture. Anerobic plate-cultures unsatisfactory because dried out too rapidly at incubator temperature. Most satisfactory media have been phosphate agar, with addition of about 25 percent of ascitic fluid, and semi-coagulated horse-serum containing sodium nucleinate. Has been impossible to grow typical Vincent spirochetes in pure culture. Greatest difficulties are elimination of associated streptococci and fusiform bacilli, and extreme lability of spirochetes. Injection of 1 cc. of mixed culture on Noguchi medium, into groins of two guinea pigs, produced foul smelling subcutaneous abscess on thirteenth day. Smears therefrom showed streptococci, fusiform bacilli, and a few spirochetes.

Review of literature fails to show any successful attempt to cultivate coarse spirochete associated with Plaut-Vincent infections. This

corresponds with experience of Jahnel, who recently expressed belief that all past cultures of *Spirocheta pallida* probably saprophytic spirochetes. Determination of pathogenicity of Plaut-Vincent group of organisms must await successful cultivation of coarse spirochetes. Experimental productions of Plaut-Vincent stomatitis, without use of pure cultures of both fusiform bacillus and coarse spirochete, represent merely prototype of clinical forms of stomatitis in acute blood dyscrasias, avitaminoses, and metallic poisoning, due to marked lowering of general bodily resistance.

43. COMPARATIVE STUDIES OF LACTOBACILLUS ACIDOPHILUS ASSOCIATED WITH CARIES. *Harry P. Ulicny, B.Sc. in Phar., M.A., College of Pharmacy, Ohio State University, Columbus, Ohio. L. acidophilus* from caries studied comparatively, in biochemical reactions, with known intestinal strains. Iodometric determinations of sugar in cultural medium (Style, Peter, and Fred) used to compare sugar utilization. Results indicated dental and intestinal strains of *L. acidophilus* not of same species. Dental strains much more saccharolytic. There is closer relationship among strains of *Lactobacillus* in dental group than in intestinal. In attempt to isolate bacteriophage against various species of *L. acidophilus* of dental origin, saliva from carious individuals, peritoneal exudate from guinea pig previously inoculated with *Lactobacillus* organisms, and sewage, gave negative results. Potent bacteriophage against *Eberthella typhi* bacillus could not be adapted to lysis of caries organisms. These failures to develop "phage" might have been due to acidity of culture media for *L. acidophilus*. Calcium carbonate added to broth medium reduced acidity only temporarily. If suitable method of reducing acidity could be devised, "phage" might be developed. (To be published in *J. Den. Res.*)

44. DENTAL CARIES. I: NEW METHOD OF STUDYING BACTERIAL PLAQUE. *J. R. Blayney, B.S., M.S., D.D.S., Robert G. Kesel, M.S., D.D.S., and Edward C. Wach, Ph.G., D.D.S., College of Dentistry, University of Illinois, Chicago, Ill.* In preparing many ground sections of human teeth, bacterial plaques on enamel surface were frequently observed, as demonstrated by Williams. Beneath some plaques there was caries of enamel; beneath others, all enamel was normal.

This showed some bacterial plaques do not produce caries, and suggested hypothesis that types of contained organisms may be different. To study morphology of organisms in plaques, direct smear-preparations were made after all superficial debris removed. Carious plaque always contained either short parallel rods or coccobacilli, described by Bunting as *B. acidophilus*, with other associated forms. Non-carious plaques contained any form except typical short rods and coccobacilli, frequently including spirochetes, not associated with carious plaques.

Susceptibility or immunity to dental caries in 30 young patients studied by two methods: (a) direct smears, and (b) cultures of stimulated saliva for *B. acidophilus*, using Bunting's technic. Comparing results with both methods: agreement in 83 percent of cases. In another study of 62 children, nine years old or younger—using direct smears and culturing material from smears before fixation for *B. acidophilus*—200 negative smears gave negative cultures; 8 positive smears gave positive cultures; 7 positive smears gave negative cultures; 5 negative smears gave positive cultures. In 37 cases, in which mouth-washings were cultured—in addition to above—there were 4 positive washings, with negative smears and smear cultures; 6 negative washings with positive smears and smear cultures; 22 negative washings with negative smears and smear cultures; and 5 positive washings with positive smears and smear cultures. (See abstract 45.)

45. DENTAL CARIES. II: CLINICAL EVALUATION OF METHOD. *Harold J. Noyes, Ph.B., B.S., D.D.S., M.D., College of Dentistry, University of Illinois, Chicago, Ill.* To check diagnostic significance of smear method [abstract 44], 409 smears were taken from 64 orthodontic patients. Clinical conditions of tooth surfaces from which material was removed, and of whole mouth, recorded. Smears reported with respect to carious process at particular areas, without reference to clinical examination. Reports were analyzed from standpoint of whole mouth, by dividing patients into four groups determined by degree of susceptibility to caries, based upon complete mouth examination, in which treated and untreated carious lesions were charted; full mouth x-rays; operator's impression of activity of the disease. Results summarized on following page.

Classification	Cases No.	Lesions Av. no.	Smears No.	Smears Positive %	Smears Negative %	Examinations All positive smears %	Examinations All negative smears %
Immune	10	0	53	7.5	92.5	0	70.0
Relatively immune	24	4.1	151	13.9	86.1	0	46.6
Relatively susceptible	24	10.2	156	35.3	64.7	6.8	17.2
Susceptible	6	19.3	49	81.6	18.4	62.5	12.5

Analysis of smear reports, with respect to clinical condition of tooth surface from which scrapings were taken, revealed following correlation:

Clinical condition of tooth surface	Smears Positive %	Smears Negative %	Examinations All positive smears %	Examinations All negative smears %
Positive	73.1	26.9	59.3	18.7
Negative	13.3	86.7	1.7	71.4

Results indicated positive correlation between bacterial flora of smear, and clinical condition of caries, of both mouth as a whole and particular tooth surfaces. Diagnostic criteria upon which smear was reported appeared to be more accurate in early than in late carious lesions.

46. INCIDENCE OF FETOR EX ORE. *Charles W. Freeman, M.S., D.D.S., and Glenn F. Sulser, D.D.S., Dental School, Northwestern University, Chicago, Ill.* Because of lack of accurate data on incidence of *fetor ex ore*, 1000 cases were provisionally classified. Variation in interpretation of results showed need for better classification. After several tests with odors from different compounds, following classification adopted: (a) neutral, (b) foul, (c) sour, (d) alcohol, (e) tobacco, (f) condiments, including all extraneous odors. Another 1000 cases examined on this basis; also odor interpretations recorded by these age-groups: youthful, to 16; adult, 17 to 50; older, above 50. Percentage results, in terms of above classification: neutral, 34.5; foul, 20.4; sour, 15.1; alcohol, 5.6; tobacco, 15.8; condiments, 8.6. Foul and sour breaths become more marked as age increases. Percentage of neutral breaths in youthful group, 62.1; in adult group, 28.3; in older group, 20.6. Further work in progress.

47. RATE OF GROWTH OF ALVEOLAR BONE MEASURED BY ALIZARIN INJECTIONS. *I. Schour, B.S., M.S., D.D.S., Ph.D., College of Dentistry, University of Illinois, Chicago, Ill.* Each of sixteen white rats, about three months old, given two intraperitoneal injections of 0.5 cc. of 2 percent alizarin red S (sodium sulphalizarate; color index 1034; Coleman and Bell Co.) four or six days apart, and killed two days after last injection. Dosage ranged from 39 to 62 mgm. per k. Microscopic examination of sections ground in oil showed red lines in portions of dentin, cementum, and in alveolar and long bones calcifying at times of injection. Amount of bone laid down in 24 hours calculated by measuring, in μ, vertical distance between two red lines, and dividing by number of days between injections. Alizarin lines more regular in alveolar bone of incisors and molars than in long bones. Total of 471 measurements, independently by three observers, showed average daily apposition of alveolar bone was 4 μ—approximately fourth the daily apposition (16 μ) of enamel and dentin in incisors. Measurements in alveolar bone of teeth of *Macacus rhesus* monkeys, given intraperitoneal or intravenous injections of sodium sulphalizarate, indicated much slower apposition; this phase of study not yet completed. Multiple injections of sodium sulphalizarate offer prompt and accurate method of measuring rate of apposition of bone in normal and experimental animals.

48. CLINICAL EXPERIENCE IN APPLICATION OF ULTRA-SHORT ELECTRIC WAVES IN DENTISTRY. *J. S. Oartel, D.D.S., M.S., F.A.C.D., Dental School, University of Pittsburgh, Pittsburgh, Pa.* Author previously reported lethal effects on streptococci in dentin of extracted infected teeth after exposure to short electric waves. Present study determined whether lethal effects on microörganisms could be produced in infected teeth in mouth. Infected roentgen-positive teeth treated with ultra-short-wave therapy-machine having wave-length of six meters; cultures of root contents made before, and at intervals during, treatment. After five to seven treatments, cultures negative. Bone regeneration about apices observed in eight weeks after beginning of treatment. Ultra-short-wave therapy distinctly beneficial in cases of acute alveolar abscess, dry socket, infections of frontal and maxillary sinuses, and osteomyelitis.

49. AGE INCIDENCE OF CARIES. *Mary M. Moore, D.D.S., Philadel-*

phia, Pa. Past reports of efforts to control caries based on observations of children not over 14 years old. Author's experience led to question assumption that little or no caries before 14 assures little or none thereafter. Report covers 80 cases seen on average of three or four times a year over age-period 6 to 21. All children had not only adequate dental care but also benefit of medical care and good food. In recording cavities, occlusal surfaces not counted. In author's practice all treated as potential or apparent caries at time of eruption. Peak years, both for group having caries and for number of cavities, were 17, 18, and 19. This spread of peak corresponds with individual differences between physiologic and chronologic ages. There was 20.6 percent of total decay from ages 6 to 13; 79.5 from 14 to 21—146 percent more decay between 14 and 21 than would have resulted if rate of decay from 6 to 13 had continued through to 21. Middle and later years of adolescence is period of greatest decay. Unless observations are made during this period, and evidence of control of decay given, it cannot be safely assumed that any then existing factor or group of factors prevents caries. (Published in *Ann. of Den.*, 1936, *2*, 77; June.)

IX. Fifth Session: Afternoon, March 15; Abstracts 50–63

50. Effects of subcutaneous injections of oestrogenic and gonadotropic hormones on gums and oral mucous membranes of normal and castrated rhesus monkeys. *Daniel E. Ziskin, D.D.S., S. N. Blackberg, Ph.D., and C. A. Slanetz, B.S., M.S., Ph.D., Dental and Medical Schools, Columbia University, New York City.* Lesions resembling changes in so-called "pregnancy gingivitis" (swelling and old-rose color of interdental papillae) induced in normal rhesus monkeys by injection of gonadotropic hormone extracted from human pregnancy-urine (PU). In ovariectomized monkeys injected with gonadotropic hormone (Folluetin-Squibb; a refinement of previously used extract of pregnancy-urine), gums acquired clinical appearance of Vincent infection; tendency to easy bleeding; swelling of interdental papillae; later, necrosis along crests. With injection into ovariectomized monkeys of estrogenic hormone (Amniotin-Squibb, or Progynon B), gum tissue appeared firmer and better able to resist infection than normally. Results similar to those produced with

Amniotin followed injections into normal monkeys of two gonadotropic hormones—extract of anterior pituitary gland of sheep (AP) and extract of urine of castrated women (CU). Two methods of *control* utilized: pre-experimental tissue obtained whenever possible; tissue of untreated castrated female monkeys also taken; changes noted above not observed. *Conclusions*: Oestrogenic and gonadotropic hormones cause changes in gums and oral mucous-membranes of normal and castrated rhesus monkeys. Changes with PU in normal monkeys resembled so-called "pregnancy gingivitis" (by us termed "hormonal gingivitis"); in castrated group their character was that of Vincent infection. However, Amniotin, Progynon B, AP, and CU apparently strengthen gums, probably owing to increased cellular activity and rapid production of keratin. (Histologic findings to be detailed in complete paper; to be published in *J. Den. Res.*)

51. HISTOPATHOLOGY OF TEETH OF GUINEA PIGS IN ACUTE AND CHRONIC VITAMIN-C DEFICIENCY. *P. E. Boyle, D.M.D., S. B. Wolbach, M.D., and O. A. Bessey, Ph.D., Dental and Medical Schools, Harvard University, Boston, Mass.* Guinea pigs have been kept on vitamin-C-free diet plus measured amounts of Cebione (Merck) varying from 0.3 to 2.0 mgm., daily. Negative control groups on basal diet alone, and positive control groups on larger amounts of ascorbic acid and on greens, included. Previous report (*Arch. Path.*, 1926, *1*, 1) emphasized, as fundamental phenomenon of scurvy, inability of certain cells of mesenchymal origin to produce intercellular material. Present study confirms previous findings. In acute scurvy (basal diet alone), marked atrophy of cells of dental pulp occurred. Odontoblasts became morphologically indistinguishable from other pulp cells. Dentin deposition ceased. Previously deposited predentin heavily calcified. On 0.3-mgm. level of ascorbic-acid administration, animals remained alive eight to ten weeks. Tooth formation continued normally (approximately 2 mm. per week). Mantle dentin, deposited at basal ends of incisors, appeared normal. No discernible change in odontoblasts in this region. Farther incisally, odontoblasts assumed papillary arrangement about capillaries situated pulpally. Normal capillary plexus about dental portion of odontoblast absent. Width of dentin, including predentin, less than width of enamel. Still farther apically, long spicules of dentin projected into pulp

cavity. Hemorrhages present in enamel organ and periodontal membrane. Occasional enamel hypoplasias noted. Minute fractures of alveolar bone occurred frequently. Tooth structure formed during experimental period, on eruption into oral cavity, extremely fragile. Similar but less marked changes in animals on 0.5 and 1.0 mgm. per day; on these levels, survived for long periods. The 2.0-mgm. level maintained animals in approximately normal condition. Guinea pig evidently able to adapt itself to low level of vitamin-C intake.

52. PROPOSED FEDERAL SPECIFICATIONS FOR TOOTH PASTE. *Wilmer Souder, Ph.D., F.A.C.D., and I. C. Schoonover, Ph.D., National Bureau of Standards, Washington, D. C.* (*a*) Shall assist in removal of daily accumulation of debris from exposed surfaces of teeth without injury. (*b*) Shall not injure mucous membrane of mouth. (*c*) Shall not segregate, ferment, or deteriorate at 5°F. for 1 hour; or at 122°F. for 72 hours. (*d*) Forty (40) percent or more shall be from calcium carbonate, di- or tri-calcium phosphate, magnesium carbonate, magnesium hydroxide. (*e*) Shall not be more than 20 percent soap, 2.5 percent sodium benzoate. (*f*) May contain sodium bicarbonate, sodium borate, sodium chloride, boric acid, citric acid, sugars, saccharin, acetic acid, cream of tartar, gelatin, gums, mineral oil, alcohol, glycerin, water, flavors, and colors that bear approval of U. S. Dept. of Agriculture, Food and Drug Administration. (*g*) Shall not contain poisons when tested according to U.S.P. XI methods. (*h*) Ninety-eight (98) percent shall pass no.-200 sieve. (*i*) pH shall be between 5 and 10. (*j*) Shall not scratch glass. (*k*) Formula shall be supplied with bid. Above data supplied for discussion only. There is, at this date (March 15), no Federal specification for tooth paste.

53. WORK OF RESEARCH COMMISSION OF AMERICAN DENTAL ASSOCIATION. *P. C. Lowery, D.D.S., F.A.C.D., Chairman, Detroit, Mich.* During author's membership in Research Commission, pendulum has swung from 100 percent biological research to present plan—nearly all physical research. Biological research necessarily takes longer to produce constructive results; to some members of Research Commission seems less important; but to most members, including author, both types of research necessary and important. Extensive work on dental materials now conducted by Fellows of American Dental Asso-

ciation, and members of Federal Government, at National Bureau of Standards. For such materials, standards of quality are being written, to which dental manufacturers are very receptive. Organized dentistry also stimulated and sponsored this work in dental schools, several having supplied sufficient equipment and personnel for courses on physical properties of dental materials. Already decided improvements in quality and methods of handling these materials is evident. Findings raised standards of dentistry and increased life-span of operative dentistry, particularly of gold-inlay and amalgam restorations. Attack upon dental caries well under way. Committee of three internationally known men, representing three different professions, appointed as special advisory committee on dental caries, to correlate, and place in tangible and usable form, all previous literature on dental caries—to include information from all allied professions, from all countries. Commission hopes American Dental Association will finance program whereby at least three fellowships may be created in one or more important research institutions for work on specific problems of dental caries—fellows to be selected from men well trained in one or more allied fields (engineering, biochemistry, bacteriology, pathology, immunology, eugenics, nutrition and metabolism).

54. PULPS AND INVESTING TISSUES OF EMBEDDED TEETH AND ROOT FRAGMENTS. *W. H. G. Logan, M.S., M.D., D.D.S., F.A.C.S., F.A.C.D., Foundation for Dental Research of the Chicago College of Dental Surgery, Chicago, Ill.* Difficulty in avoiding contamination, when removing teeth and other tissues from oral cavity for bacteriologic examination, requires determination of presence or absence of pathogenic microörganisms in oral and dental tissues. Methods for this purpose are histologic examination of tissues, and staining to detect bacteria within tissues. Twenty-four embedded teeth examined: 14 completely covered by intact overlying tissues; 10 had communication with oral cavity. Four of embedded teeth (2 fully embedded, 2 partially erupted) examined *in situ* within jaws obtained by autopsy. Bacteria found on surface of all embedded teeth accessible to oral fluids; also in superficial portion of periodontal membrane. For fully embedded teeth, neither bacteria nor inflammatory tissue-reactions in adjacent structures. For none of 24 embedded teeth so

far examined were microörganisms or inflammatory changes found in pulps. Several pulps of embedded teeth showed calcifications (pulp stones); similar pulp stones present in some erupted teeth in same mouth.

55. VERIFICATION OF ROOT-CANAL TREATMENT IN DOGS. *C. M. Dixon, D.D.S., and U. Garfield Rickert, A.M., D.D.S., F.A.C.D., Dental School, University of Michigan, Ann Arbor, Mich.* Experimental animals were used to study tissue reactions in two types of root-canal cases: infected cases and so-called "vital" ones. Infected cases initiated by inoculating teeth (pulps removed) with 24-hour culture of *Str. viridans* from infected teeth of clinic patients. Vital cases treated as such; infected cases, after showing roentgenographic evidence of alveolar change (parodontitis). Treatments consisted of medications with formocresol until two sterile cultures obtained, followed by filling with Kerr Sealer and gutta-percha according to Rickert technic (used in School Clinic). Vital cases treated by milder medication, but filled by same method after obtaining negative cultures. Histologically, infected cases showed healing by production of secondary cementum and regeneration of periodontal membrane, about apices and within lateral canals. Vital cases showed no alveolar destruction, not having been infected; there were normal periodontal membrane and secondary cementum in some lateral canals. Teeth cultured before necropsy—and inoculum transferred to suitable culture media and incubated for seven days—showed only one growth: spore former of usual air-contaminant type. Duplicate cultures were obtained by Rosenow method; after 5 days, all showed spore formers and contaminants.

56. HISTO-PATHOLOGY OF AMELOBLASTOMA IN TWO AND ONE-HALF YEAR OLD CHILD. *Hamilton B. G. Robinson, D.D.S., Rockefeller Fellow in Dentistry, School of Medicine and Dentistry, University of Rochester, Rochester, N. Y.* Firm, pale, encapsulated mass removed from left mandible of 2.5-year-old child, by Dr. J. J. Morton, Surgeon-in-Chief, Strong Memorial Hospital. Growth had first been noticed six months previously. Lobulated mass weighed 52 gm.; measured 6.5 x 4 x 3 cm. Histologic section revealed mass of connective tissue of embryonal type pervaded by branching and budding strands of columnar epithelial cells, some showing tendency to cyst formation.

Many strands and buds suggested structure of dental lamina and enamel knot. In other areas, structures closely resembling more mature stage of normal enamel organ—with cells analogous to those of ameloblastic layer, stratum intermedium, and stellate reticulum —evident. This young ameloblastoma presented picture highly suggestive of origin from cells having potentiality for enamel formation. Since no connective tissue capable of dentin production was present, and as formation of that tissue must always precede development of enamel, cystic degeneration of tumor occurred after attainment of functional stage. "Epitheliom enstanden aus dem Schmelzorgan" reported in new-born child by Massin (1894), and unique, melanotic ameloblastoma in 5.5-month infant by Mummery and Pitts (1926); but present is youngest recorded case having ameloblastoma of typical structure. (Clinical report to be published by Dr. J. J. Morton in *Am. J. Cancer.*)

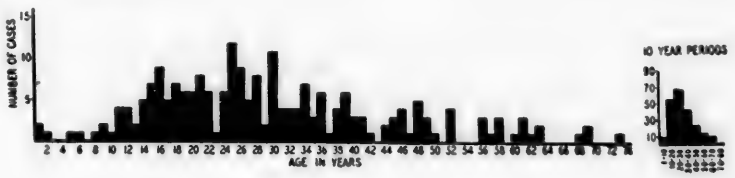

FIG. 1. Graph showing ages at discovery of ameloblastomas; 220 cases.

57. AGE-INCIDENCE STUDIES OF AMELOBLASTOMAS. *Hamilton B. G. Robinson, D.D.S., Rockefeller Fellow in Dentistry, School of Medicine and Dentistry, University of Rochester, Rochester, N. Y.* Review of literature reveals about 140 articles (English, French, German, Italian, American, medical and dental journals) containing reports on ameloblastomas. Total of 357 cases reported; in many instances, data incomplete. Of 296 ameloblastomas, 54.9 percent in females. Racial distribution could not be determined; 37 Negroes, 2 Chinese, and 2 East Indians included. Indicated ages, not uniform; some represent time of presentation—others, of operation or discharge. "Age of report" cited in 247 instances; average, 36.9 years. Duration of growth—or, more accurately, difference between "age of report" and age of discovery of growth—averaged 8.9 years for 230 cases; maximum, 53 years. Age of discovery, computed in 220

cases, averaged 29.5 years. Seventy percent first noticed between 10 and 35 years (*fig. 1*). Ameloblastomas may occur earlier than is usually assumed, and may persist very long time without becoming invasive to point of destroying hosts.

58. ACUTE MYELOGENOUS LEUKEMIA: CASE WITH SIGNIFICANT ORAL MANIFESTATIONS. *Thomas J. Cook, D.D.S., School of Dentistry, University of Pennsylvania, Philadelphia, Pa.* Following observations support opinion that severe oral sepsis is main feature. Oct. 28, 1935: acutely ill, single, white female, age 35, admitted to University Hospital. Diagnosis: acute myelogenous leukemia. At author's entrance into case, three days later, patient complained of pain on mandibular right side, and gave history of having similar pain on maxillary left side, week before. Oral examination revealed noticeable fetor of breath; full complement of teeth showing excellent care. Mandibular second premolar, first and second molars, loose. Ulceration on lingual surface. Despite previous history of pain, maxillary left molars negative. Gingivitis evident on palatal gum of maxillary incisors; all maxillary teeth firm. Smears showed numerous Vincent organisms, and staphylococci and streptococci. Dental roentgenograms of mandibular right side disclosed horizontal absorption. Impossible to make roentgenograms of entire mouth. Extraction not attempted. Progress of ulceration, and necrosis of palatal tissues, amazing. Necessary, in two weeks, to remove necrotic masses daily so patient could be fed. Before death, Nov. 30, maxillary incisor teeth became so loose were removed with necrotic tissue; remaining upper teeth freely movable by pressure of tongue. Severe necrosis of entire maxilla and right side of mandible. Case reported as bearing on published instances of blood dyscrasias where attacks and death followed extraction of teeth.

59. CHEMICAL COMPOSITION OF STIMULATED AND NON-STIMULATED SALIVAS IN RELATION TO DENTAL CARIES. *Julius White, M.A., Ph.D., and Russell W. Bunting, D.D.S., D.D.Sc., F.A.C.D., School of Dentistry, University of Michigan, Ann Arbor, Mich.* Comparative study of Ca, P, pH, CO_2-capacity, ash, and total solids, of resting and stimulated salivas (paraffin chewing) of 12 caries-free and 13 caries-susceptible children—9 to 16 years. Outgrowth of criticism by Becks and Wainwright of work of Hubbell and Bunting on ground non-stimulated

salivas were used. Data for stimulated salivas confirmed work of Hubbell and Bunting. Although there were greater amounts of Ca and P, and much lower CO_2-capacity, in resting salivas, values obtained for caries-free individuals were in same range as those for caries-susceptible. In both stimulated and non-stimulated saliva, therefore, Ca and P contents bear no relationship to caries.

60. FACTORS INFLUENCING ACID-NEUTRALIZING POWER OF SALIVA. *J. H. Wills, Jr., B.S., and J. C. Forbes, Ph.D., Medical College of Virginia, Richmond, Va.* Effects of several common factors upon acid-neutralizing power of saliva investigated. High protein-low carbohydrate diet increased, high carbohydrate-low protein diet decreased, it. Thorough cleansing of teeth after each meal, to remove food particles: no influence. Addition of spinach to diet raised acid-neutralizing power to value above level of diet alone. In general, single *carbohydrate* meal produced marked fall in acid-neutralizing power, with gradual return to general level of diet. Single *protein* meal produced first rise and then return to dietary level. Usually maximum effect obtained about 1 hour after meal; at end of 2.5 hours, acid-neutralizing power approximated original level. Neutralizing power, after chewing sponge rubber, chewing gum, or paraffin, for five minutes, generally increased, but elevation not constant; in two cases, decrease followed gum chewing. Smoking, and cleaning teeth, produced very transient elevations of acid-neutralizing power. (To be published in *J. Den. Res.*)

61. MOUSE-PROTECTION TESTS WITH SALIVA. *J. L. T. Appleton, B.S., D.D.S., Sc.D., School of Dentistry, University of Pennsylvania, Philadelphia, Pa.* Following principle of mouse-protection tests with antipneumococcic sera, attempt was made to determine if human centrifuged saliva exerted similar effect. Data obtained with unstimulated saliva of one individual. Similar, but fewer, tests with salivas of other individuals gave corroboratory results. Bacterium used: *Serratia marcescens*. Three sets of mice: (*a*) received intraperitoneally 0.5 cc. of centrifuged saliva alone—only one death occurred in this group; (*b*) received intraperitoneally 0.5 cc. of 24-hour broth-culture of bacterium, diluted 1:7 with sterile broth; (*c*) received intraperitoneally 0.5 cc. of 24-hour broth-culture of bacterium, diluted 1:7 with centrifuged saliva. After injection, animals (more

than 200) observed for 72 hours. Deaths and their order noted. Heart-blood cultures taken from animals that died; only two or three failed to yield bacterium injected—these discarded in calculating results. After 72 hours, all survivors chloroformed and heart-blood cultures taken. There were (*a*) lower gross mortality, (*b*) postponement of death, and (*c*) fewer positive, heart-blood cultures among survivors, in mice receiving saliva suspensions than in those receiving broth suspensions. This work in progress with other bacteria, including a pneumococcus, in place of *S. marcescens*.

62. WILLIAMS' CLASSIFICATION OF TOOTH FORMS. *M. Russell Stein, D.D.S., Dental School, Columbia University, New York City.* Williams devised classification of anterior crown-forms primarily as commercial enterprise, and intended it should replace "racial" and "temperament" classifications. His three chief arguments against old theories: (*a*) there is no correlation between racial type and tooth form; (*b*) no correlation between form of cranium or face and shape of teeth; and (*c*) no demonstrable connection between shape of central incisors and temperament. He claimed that three fundamental types of his classification were "natural," and showed they existed in apes, using this fact as another proof of human-anthropoid affinity. Variability in outlines of central incisors, however, is so great many geometric classifications are possible, Williams' three types being neither natural nor fundamental. When his own examples of Class II are superimposed upon Class III: practically no difference. Among many inconsistencies in his thesis is fact that he argued for harmony in face and tooth forms in selection of artificial teeth just after he denied existence of such harmony in disposing of rival theories. (Published in *J. Am. Den. Assoc.*, 1936, *23*, 1512.)

63. ROUND-CELL SARCOMA OF MANDIBLE. *Paul C. Kitchin, M.S., D.D.S., Dental School, and Charles A. Doan, M.D., Medical School, Ohio State University, Columbus, Ohio.* Case of mandibular round-cell sarcoma with illustrations of oral condition; photomicrographs of biopsy material; reaction of lesions to massive doses of deep x-ray therapy; oral roentgenograms of tumor-involved areas; hematologic determinations during course of disease and treatment. Necessity for careful study of roentgenograms in every case of loose teeth, and for blood determinations and biopsy examinations for cases in which

films show variation from usual picture of root abscess or periodontoclasia, emphasized. (To be published in *J. Den. Res.*)

X. Sixth Session: Evening, March 15; Abstracts 64–74

A. Greetings from representatives of dental organizations: abstracts 64–71

64. University of Louisville. *Raymond A. Kent, A.M., Ph.D., President.* President Kent welcomed the International Association for Dental Research in the name of the University of Louisville and of the City of Louisville. He expressed appreciation for the work which the Association is doing, and stated that the first reference he had ever heard made to the University of Louisville was in 1924—to the research then being carried on by Dr. Theodore Beust, the present President of the Association. He stated further that he was happy to have this opportunity to acknowledge an obligation which he had for years sustained toward the man who is chiefly responsible for the existence of the Association. He said that twelve years ago he "had the pleasure of being associated with this gentleman on committee work at another university, and was deeply impressed with the patience, the scholarliness, the thoroughness, and with all the high quality of gentlemanliness which Dr. Gies showed." These characteristics had so impressed him as not only to remain with him during the intervening years, but "in the meanwhile to have crystallized into incentives and ideals of work in the field of his own labors." It was, he said, therefore a double privilege that he had in welcoming the Association—a personal one as well as a professional one.—[J. T. O'R.]

65. Louisville district dental society. *G. M. Edwards, D.D.S., President.* "We are glad to have you as guest of our Society as well as guest of our City: because of whom you are and the cause you represent; also because of the work you are doing, even among us as well as elsewhere. We are also delighted to have you as our guest for another reason: four members of your group are members of the Louisville District Dental Society and residents of Louisville. Last, but not least, I have personal pride in extending to you a warm and cordial welcome for the fact that one of my former instructors, while I was attending the University of Louisville, is none other than your active President, Dr. Theodore Beust."

66. UNIVERSITY OF LOUISVILLE, SCHOOL OF DENTISTRY. *John T. O'Rourke, D.D.S., F.A.C.D., Dean.* It is indeed a pleasure to welcome the International Association for Dental Research, and to express the gratitude of the faculty and student-body of the School of Dentistry for the inspiration they have gained from this meeting. Fortunate is any individual who has the opportunity to attend the meetings of this Association, for its activities not only indicate trends and progress in research, but also carry the elements of stimulation and inspiration that are so necessary to progress in all fields of endeavor. While attending these meetings, one is impressed by the splendid growth of the Association; by the large amount of new knowledge contributed by its members; and by the tremendous scope of fields yet unexplored. Certainly what knowledge we now possess is insignificant in comparison to that which we must have before many current dental and related problems can be solved. The Association, therefore, is making significant contributions to human welfare and to dentistry. I am certain that it will grow rapidly in importance and in membership, and, through the stimulation it offers, cause a further marked rise in dental research in this country and abroad.

67. KENTUCKY STATE DENTAL ASSOCIATION. *J. H. Gunkel, D.D.S., President, Newport, Ky.* The speaker, referring to the honor and pleasure of addressing the Association, said that, in his official capacity, he thanked the Association for deciding to hold this meeting in Kentucky, and complimented the Association "for the wonderful work you have accomplished for dentistry in the past and promise to achieve in the future." He stressed the importance of organization. The dental profession is composed of all grades of dentists, the average of which is always less than the best. The way to raise the average is not to decrease the responsibility of the individual, but to increase it—to educate the least efficient to the level of the most efficient. To attain this objective, dental societies have been formed, which now represent professional opinions more effectively than ever.

68. AMERICAN DENTAL ASSOCIATION. *L. M. S. Miner, M.D., D.M.D., Sc.D., F.A.C.D., President-elect; Dean, Dental School, Harvard University, Boston, Mass.* Observing this large and enthusiastic group, all interested in promoting the welfare of dental research, my mind goes back to the early meetings of this organization, when this dinner-

occasion on a Sunday night consisted of a small round table, holding ten or twelve men, earnest and eager, and even militant, for the interests of dental research. In comparing those meetings with this large one, it is both gratifying and inspiring to see how the seed of dental research, planted in this organization years ago, has now grown to such a sizeable plant. I have been asked to speak for the American Dental Association. I am happy to have the honor of bringing to this organization the greetings of the officers and the nearly 40,000 members of the American Dental Association, which, for a long time, has been interested in promoting dental research. As you know, it has a Research Commission, and during the last ten years, has expended many thousands of dollars in supporting dental research. A very important matter has been before the International Association for Dental Research for the last two years; namely, the *Journal of Dental Research.* The very effective and constructive work Dr. Gies has done for dentistry through this *Journal* needs no praise, or even comment, from me. Paraphrasing the words of a Massachusetts patriot: "There it stands." But Dr. Gies has made it plain that some changes should be made, and the American Dental Association has been approached regarding the help it might give to the *Journal of Dental Research.* Personally, I feel that the happiest solution of the problem would be the continuance of the *Journal,* under the auspices of the International Association for Dental Research, but subsidized through a grant from the American Dental Association. I think the American Dental Association could further the interests of dental research effectively in this way. I shall be happy to use what influence I might have to bring this about, if it meets with the approval of the members of the International Association for Dental Research. I congratulate President Beust and Secretary Gies, for the excellent program of this meeting, and for the very fine way all matters have been conducted.

69. AMERICAN ASSOCIATION OF DENTAL SCHOOLS. *A. R. McDowell, D.D.S., F.A.C.D., President; Dean, Dental School, College of Physicians and Surgeons, San Francisco, Calif.* It is my privilege and pleasure, on this delightful and inspiring occasion, to extend felicitations of the executives and teachers of the dental schools of America, with their best wishes for the continued success of the ideals, objectives, and

purposes of your very necessary, useful, and influential Association. You are cordially invited to attend the three-day sessions of the School Association, commencing tomorrow morning in this hotel. The reciprocal relations existing between the International Association for Dental Research and the American Association of Dental Schools are most gratifying. I hope this cordial coöperation will long endure. Dental research and dental education are twins. Neither is complete without the other, and each strives to advance the profession and to make it more useful in its public service. A great difference is noticeable between the qualities of dental education in schools where research forms the basic underlying principle of instruction and in schools in which there is no research. Without research empiricism prevails, and teaching is inert, lifeless, and dead; with research, instruction is rational, vigorous, and animated. Teachers, students, and institutions are greatly affected and benefited by the enlightenment from research, especially when the attitude of the investigators is modest, humble, and tolerant. Dental schools are attaining a better understanding of research over that of twenty, fifteen, or even five years ago. With this understanding has come improved teaching. Students enjoy most those teachers who are working on fact, not dogma. The report of the Curriculum Survey Committee strongly emphasizes the need for biological study in dental behavior. Your Association has done and is doing excellent work in the promotion of research in dentistry. The *Journal of Dental Research* is serving a most useful purpose in reporting the results of investigators, teachers, and others who are interested in the discovery of new truths. You are to be congratulated on your Association's attainment of direct affiliation with the American Association for the Advancement of Science, with a member (Dr. Thomas J. Hill) on the Council of that outstanding scientific organization.

70. OMICRON KAPPA UPSILON. *R. S. Vinsant, A.B., D.D.S., F.A.C.D., President, Memphis, Tenn.* I bring you greetings from 4,500 members of the dental profession in the United States who were outstanding students in their dental courses, or have made real contributions to dental progress. We congratulate you upon your accomplishments, and thank you for the fine coöperation you have given the dental profession. You have helped us to grow from a trade

into a scientific profession. Twenty years ago, I heard the President of a state university advise a young man who had received the B.A. degree not to go into the study of dentistry. He said dentistry was a trade union, and that the young man would be wasting his time to study it. A short time ago, I heard the President of the same state university say that dentistry had made more progress in the last decade than any of the professions. Your Association has done more to give dentistry its recognition among the scientific professions than any organization bearing the name of dentistry. If I were a military man, I would say that your organization is dentistry's strongest fort. You are the Gibraltar upon which the inland sea of dentistry can depend, should war be declared upon us. If I were a naturalist, I would name this body the latent energy in dentistry, with a force sufficient to carry out its great ideals. If I were an engineer, I would say that you are the horse-power in the engine, which will enable dentistry to make the steep grade which the profession has ahead of it in the twentieth century. Dentistry is much indebted to your Secretary, Dr. Gies. A few years ago he made a study of dentistry in all of its various phases. He found our weak spots. He did not stop there, but set out to help us solve the problems which he found. While not a dentist, he has done more for dentistry than we can ever know, in bringing together the bodies which form the American Association of Dental Schools; in constructive leadership in the Dental Educational Council; in stimulating activities in the American College of Dentists; in the establishment of the American Association of Dental Editors; and in the organization of the International Association for Dental Research. The real test of any organized group is whether they hand on, in an improved condition, to posterity the civilization they inherited. You have already passed the test. This organization has stimulated research in dental problems in all parts of the world. Where there was one research worker in dentistry twenty years ago, there are fifty today. May this progress continue.

71. ACTIVITIES OF THE DENTAL GROUP AT THE NATIONAL BUREAU OF STANDARDS. *Clinton T. Messner, D.D.S., F.A.C.D., Secretary of the Research Commission of the American Dental Association; U. S. Public Health Service, Washington, D. C.* Research methods at the National Bureau of Standards are steadily improving the standards

for the manufacture of dental materials. The cumulative current influence of this work on manufacturers was stressed. Adherence by manufacturers to specifications established by the Bureau for amalgams, for example, had risen from 20 percent in 1920 to practically all well-known brands now on the market. The speaker lauded the spirit of the Research Commission, which had aroused interest in research among dental practitioners.—[T. B. B.]

B. OFFICIAL ADDRESSES: 72–74

72. PRESIDENTIAL ADDRESS. *Theodore B. Beust, D.D.S., M.D., F.A.C.D., Dental School, University of Louisville, Louisville, Ky.* (Pages 296–299.)

73. INTRODUCTION OF PRESIDENT-ELECT, BY *Isaac Schour, B.S., D.D.S., M.S., Ph.D., Dental School, University of Illinois, Chicago, Ill.* (Pages 299–301.)

74. INAUGURAL ADDRESS. *William G. Skillen, D.D.S., Dental School, Northwestern University, Chicago, Ill.* (Pages 301-304.)

[Executive proceedings followed: page 377]

XI. PAPERS READ BY TITLE: ABSTRACTS 75–120

A. CONTRIBUTIONS BY VIENNA SECTION: ABSTRACTS 75–93

75. RELATIONSHIP BETWEEN SECOND AND THIRD MOLARS. *J. Bocak, M.D., Histological Laboratory (Director Prof. Dr. B. Gottlieb), Dental Institute (Director Prof. Dr. H. Pichler), University of Vienna, Vienna, Austria.* In very large percentage of human specimens there was resorption on distal surface of second molar (covered with cementum), caused by pressure from erupting third molar. During period of eruption, crown of third molar approaches root surface of second, causing resorption on cementum; during subsequent eruption, resorbed surface repaired. *Fig. 2* shows portions of lower left second and third molars of 19-year-old individual. Higher magnification (*fig. 3*) shows area of interdental-bone margin, and resorption on second molar, repaired by secondary cementum. Primary cementum missing from surface; in older individuals covers surface. Resorption in second molar took place before present stage of eruption of third.

76. USE OF RUBBER WORK IN ORTHODONTICS. *Karl Breitner, M.D.,*

FIG. 2

FIG. 3

FIG. 4

FIG. 5

FIG. 2. Lower left second and third molars; 19 years. See *fig. 3.*

FIG. 3. Higher magnification of interdental region in *fig. 2.*

FIG. 4. Tooth root showing effects of inflammation, with deposit of cementum at apex, extending into canal. See *fig. 5.*

FIG. 5. Higher magnification of part of *fig. 4.*

FIG. 6

FIG. 7

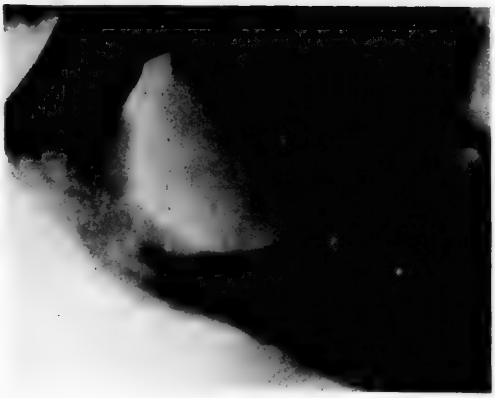

FIG. 8

FIG. 9

FIG. 10

FIG. 6. Adamantinoma *before* first operation.
FIG. 7. Adamantinoma *after* first operation.
FIG. 8. Profile picture of lower jaw during healing stage, *after* second operation.
FIG. 9. Apex of third molar (age, 36), showing necrosis at *a*. See *fig. 10*.
FIG. 10. Higher magnification of cartilaginous tissue shown in *fig. 9*.

Vienna, Austria. In orthodontic therapeutics various shapes of rubber-plate again in great demand. Can be produced easily and inexpensively; few orthodontic cases in which rubber-plate cannot be used advantageously at some stage of treatment. Orthodontic rubber

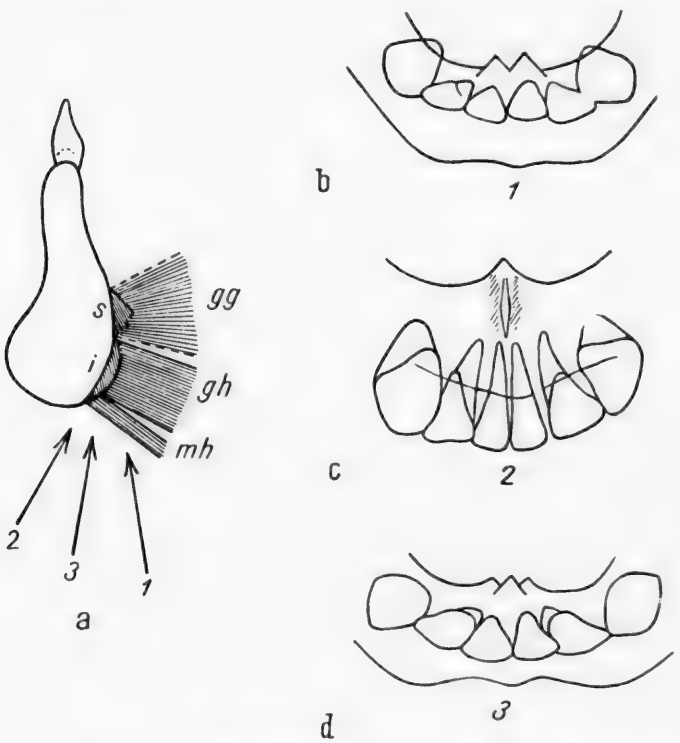

FIG. 11. CROSS SECTION AND X-RAY OUTLINES OF CHIN

a—Median sagittal section through chin bone. *s*, pars superior spinae. *i*, pars inferior spinae. *gg*, musculus genioglossus. *gh*, musculus geniohyoideus. *mh*, musculus mylohyoideus.

b, *c*, *d*.—X-ray outlines of chin, pictured from below; showing three views of spina mentalis, along lines (arrows) 1, 2, and 3, respectively, in *a*.

apparatus can be used for both active treatment and retention (support). For *active* treatment: palate plate in upper jaw, with "Aufbiss" (bite); for lower front, to shorten it; rubber-plate can be given inclined planes, to influence teeth of opposite jaw; false indentation

in region of molars removable through temporary lock-jaw with splints of rubber; divided rubber-plates applicable for widening jaws, especially with deciduous dentition, when germs of permanent teeth may be influenced in right way. Rubber plates are available for effective *retention* of attained movements of teeth; which, being much more frequently centrifugal than otherwise, are sufficiently retained by plate that fits inside mouth and upper jaw. If, in addition, plates have suitable clasps, so they cling to teeth, rubber plates can also be

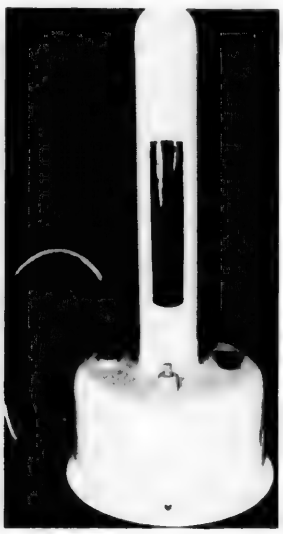

FIG. 12. Sternax controller, to measure coulombs and m. a. min.

used effectually as points of attack for intermaxillary forces when correcting false ("Kieferbeziehungen") Classes II and III.

77. TREATMENT OF HYPERSENSITIVE DENTIN. *Fritz Driak, M.D., Dental Institute (Director Prof. Dr. H. Pichler), University of Vienna, Vienna, Austria.* Dentinal hypersensitiveness may occur in hypoplasia, erosion, abrasion, caries, after injury, and during dental treatment. Cause not yet fully determined, but pain depends on position of sensitive locus and individual reaction time ("Reizschwelle"). Means for desensitization include use of (*a*) sharp instruments; (*b*) caustic and drying medicaments; (*c*) local and (*d*) general anesthetics.

Sharp hand-instruments and sharp burs, used with celerity, common expedients. Chemical agents include alkalies, acids, heavy-metal salts, alkali unguents and mouth-washes, chlorophenol, carbolic acid, formalin. Silver nitrate and zinc chloride especially effective. Silver nitrate solution (5–30 percent), applied on teeth prepared for crowns, then precipitated with hydrochinon (5 percent), very efficient as protective bandage. Local anesthetics include cocain and substitutes, and novocain (infiltrations) and derivatives. Strong effect obtained with so-called "pressure anesthesia." Cataphoresis, Black's "electrical cocain application," another procedure. Pulp lesions apt to be caused by various anesthesizing pastes. Drastic methods are cold anesthesias—act physically. Simplest way to obtain anesthesia: ether gauze on dentin, or sprayed with ethyl chloride, liquid air, liquid carbonic acid, or liquid nitrous oxyde—effective for short periods only. Gazotherm demonstrated in 1920 by Fabret, yields better results (*J. Den. Res.*, 1931, *11*, 502; June). By gradually progressing refrigeration with oxygen, considerable reduction in sensitivity of dentin obtainable. For complete anesthesia in very sensitive and nervous organs (pulp, Tomes fibrils), more highly concentrated novocain solution required than for tooth extraction. Good method to prepare Class-V cavities in upper incisors: put, in lower nose, cotton pledgets infiltrated with cocain, psicain, or plenocain (anesthesia of nervous naso-palatinus scarpae). In some difficult cases, may be desirable to effect narcosis with nitrous oxide, ethyl chloride, "gewaethyl," or ether; in exceptional instances, with intravenous injection-narcosis (pernokton, evipan), or with avertin narcosis (rectal); serious dangers in these methods contraindicate common use in dentistry. Often patient, before treatment, may be given narcotic and antipyretic powders, usually per os.

78. APPOSITION OF CEMENTUM IN ROOT CANAL, AT APEX, AFTER ROOT-CANAL TREATMENT. *Bernhard Gottlieb, M.D., Histological Laboratory, Dental Institute (Director Prof. Dr. H. Pichler), University of Vienna, Vienna, Austria.* In discussions of root-canal treatment, several cases have been published by different authors, (*a*) showing apposition of cementum around apex into root canal, and (*b*) favorably interpreted and described as presenting ideal to be attained. During experimental work on dog teeth, several cases found in which, *despite*

severe inflammation, cementum deposited around apex extending into root canal (*figs. 4* and *5*). This condition in extracted teeth should be interpreted with caution. (See *figs.* on p. 345.)

79. SINUSES IN EDENTULOUS JAW. *Richard Grohs, M.D., Histological Laboratory (Director Prof. Dr. B. Gottlieb), Dental Institute (Director Prof. Dr. H. Pichler), University of Vienna, Vienna, Austria.* Upper jaw of man, 34; ten different sinuses, including incompletely eliminated cysts, and solid epithelial cord as remnant of granuloma after extraction. Lateral (paradental) sinus opened into gingival pocket. Sinus formation described as due to acute abscess and necrotic bone sequester.

80. OCCUPATIONAL DISEASE IN WORKERS OF MOTHER-OF-PEARL. *Otto Hofer, M.D., Jaw Department (Director Prof. Dr. H. Pichler), First Surgical Clinic (Director Prof. Dr. E. Ranzi), University of Vienna, Vienna, Austria.* In 1869, Englisch, first noting persons working with mother-of-pearl liable to acquire peculiar osteitis distinguished by clinical features and specific course, reported three cases of inflammatory changes in young mother-of-pearl turners, particularly of jaw and other tubular bones, associated with piercing pain and swelling of surrounding soft tissues. Affected region: usually border between epiphysis and diaphysis of tubular bone; joint unaffected. Osteitis—very hard, painful thickening—spread over inferior maxillary bone, proceeding in spindle-shaped manner throughout lower jaw. Severe ankylosis always one of symptoms; no purulence, no necrosis of bone. Inflammation extended quickly one side to other; occurred chiefly during puberty. Relapses: when patients resumed work. Etiology obscure; probably pathologic disturbances of second phase of ossification—of fusion between diaphysis and epiphysis—caused by penetration of mother-of-pearl dust into body. Uncertain whether organic substance in shell (conchiolin), or grinding slime, had specific effects.

History of present case. Feb., 1934: chief complaint of J. B. (7267), woman, 56—toothache in right lower pre-molar tooth, accompanied by slight swelling and pain to right of chin. During following eight days, swelling increased, remaining compact and hard despite intensive heat-therapy—no effect on consistency. Finally, consulting physician made external incision; no pus discharged. Few days later compact swelling appeared on left side of chin—similar to that on right—along horizontal ramus, reaching to angle of jaw. Swelling, associated with throbbing burning pain, compact; skin, intensely red.

Temperature: never much above 37°C. For month condition remained unchanged, with increasing jaw ankylosis. Simultaneously: intense gingivitis; irrigated with astringents. April: patient came for treatment. Blood-picture: 4,400,000 red cells; 1300 leucocytes; index for staining, 0.9. Differential percentage count of leucocytes: non-segmented neutrophiles, 6; segmented neutrophiles, 70; eosinophiles, 1; monocytes, 8; lymphocytes, 15; thrombocytes, many. Infiltration of edges of both sides of lower jaw considerable, extending on left to sub-maxillary region; overlying skin, reddened. Swelling slightly sensitive to pressure, and fingers left impressions in skin (edema). On right side, near region of canine, linear scar 1 cm. in length. Now ankylosis almost complete; moderate gingivitis; coating of tartar; deep pockets; slight looseness of teeth. X-ray indicated no change in structure of maxillary bone. Axial picture of lower edge of chin: sharply bordered fine stratification. No evidence of osteolysis—usually seen in osteomyelitis. X-ray showed greater Ca content in bones; spongy meshes narrower, and Ca contents greater, than usual. Since such differences in shape occur under normal conditions, cannot be considered pathognomonic of this disease. Clinical and x-ray examination of lungs: emphysema only. Wassermann reaction negative. Mental nerve, each side, no disturbance of sensation; vitality of teeth normal. Regarding specific inflammations: lues and tuberculosis excluded. Diagnosis: actinomycosis of jaw. Swelling varied frequently, being larger one side than other—no change in consistency; therefore no material removed for examination; waited for softening of swelling. KI administered internally. Treatment as outpatient with x-ray in following doses: 1934—*Apr. 7*, 2 H 10, left lower jaw. *Apr. 14*, 3 H 10, left and right lower jaw. Early April: no change of process clinically discernible. Letter from consulting family physician then drew attention to probable cause of disease—husband worked for many years in factory making mother-of-pearl buttons. No symptoms similar to those in wife occurred in factory. Owing to unfavorable economic conditions, factory closed year before; since then, husband manufacturing buttons in home with wife's aid.

Immediately after learning these facts, patient was requested to cease helping husband and, if possible, to avoid room in which he worked, to prevent injury by mother-of-pearl dust. Patient strictly followed instructions; after another x-ray irradiation of left lower jaw, with 3 H 10, came (June 9, 1934) for reëxamination. All symptoms absent; swelling as well as lock-jaw vanished; patient well. Another blood-examination showed decrease of leucocytes (720); number of red cells, 4,100,000. Differential count: practically unchanged. Fact that only this woman became ill indicates individual susceptibility. Since number of employees in mother-of-pearl factory where husband worked was large and none affected, seems probable danger of contracting disease is diminished by hygienic measures, such as ample space and good ventilation. Ailment not restricted to adolescence; present patient over 56. Treatments with KI and x-rays presumably favorable for retrogression of inflammatory conditions; but most important factor in complete healing was removal of exciting agent.

Inflammation did not cause any essential destruction of bone; had chiefly proliferative influence, as seen in x-ray films—first ever made in this occupational malady.

81. CATAPHORESIS IN ROOT-CANAL TREATMENT. *Ernst Kellner, M.D., Histological Laboratory (Director Prof. Dr. B. Gottlieb), Dental Institute (Director Prof. Dr. H. Pichler), University of Vienna, Vienna, Austria.* (No manuscript for publication.)

82. CASE OF ADAMANTINOMA. *Emerich Kotanyi, M.D., Jaw Department (Director Prof. Dr. H. Pichler), First Surgical Clinic (Director Prof. Dr. E. Ranzi), University of Vienna, Vienna, Austria.* Adamantinoma, normally, benign epithelial growth; 5 percent become malignant—grow destructively, and produce metastases. Should be kept under close observation, especially because cannot be distinguished with certainty, either clinically or by x-rays, from some dental cysts, osteitis fibrosa, myxematous fibrosa, etc. Differential diagnostic considerations not only of prognostic importance, but also determine kind of surgical intervention. Usual location is angle of jaw and ascending ramus of mandible. In this Department: no record of case of adamantinoma in *superior* maxilla.

History of present case. Two adamantinomatous cystic formations: one, around apex of tooth; other, between two teeth, root-cyst not bigger than hazel nut (*figs. 6* and *7*). Diagnosis by histological examination only; undertaken chiefly because one cyst localized between adjacent teeth. Patient: girl, 19; first seen, April 1933; since August, 1932, had tumor, buccally from right lower first molar, causing neither pain nor fever; did not diminish. Examination: fluctuating hazel-nut-sized swelling buccally from right lower first-molar root (crown destroyed by decay) and second bicuspid. Radiogram showed sharply defined radiolucent area—hazel-nut size—between roots of second lower bicuspid and first molar; two smaller, sharply defined, translucent areas in region of apex of adjacent first molar. Cyst between two teeth enucleated: although not connected with either, part of distal surface of second-bicuspid root (tested vital) denuded. Extracted first molar had granuloma—bean size—around medial apex. Histological diagnosis, for either cyst: cystic adamantinoma. Case proves diagnostic importance of histological examination of cystic membrane.

Routine for adamantinoma, this Department: removal by thorough curetting and repeated exposure to radium rays, with periodical control with x-rays (*conservative* surgical treatment, according to Prof. Pichler). This case: *radical* operation because no assurance patient would be available for future treatment. Surrounding bone and adjacent teeth removed, including healthy tissue on all surfaces, leaving sound part of lower margin of jaw, about 2 cm. in width;

no dislocation (*fig. 8*). Ordinary partial plate to replace missing teeth, prepared before operation, at once inserted to protect wound; after healing, adapted to form of jaw. *Summary*: Two small cysts, operated as root-cysts, found, on histological examination, to be cystic adamantinomata, indicating that all cystic membrane should be examined microscopically. Cystic adamantinoma of this minute size not hitherto described. (See *figs.* on p. 346.)

83. SUPERNUMERARY TEETH. *H. Mathis, M.D., Dental Institute (Director Prof. Dr. H. Pichler), University of Vienna, Vienna, Austria.* In thorough study of presence and significance of supernumerary teeth, author's findings disagree with Bolk's, especially in regard to denticle designated by Bolk as "mesiodens." Present study, primarily of x-ray pictures, facilitated determination of calcification at any given stage, and afforded reliable conclusions as to period of original formation of teeth. In most cases, original formation of "mesiodens" occurs between that of deciduous teeth and permanent teeth. "Mesiodens," belonging to order of its own, does not erupt with former because it is most mesial element in group of permanent teeth (as Bolk concluded), but instead because it is intercalary element. Formation of intercalary elements might be regarded as reminiscent of polyphyodontal conditions of ancestors.

84. CARTILAGE IN PERIODONTAL MEMBRANE. *Balint Orban, M.D., Histological Laboratory (Director Prof. Dr. B. Gottlieb), Dental Institute (Director Prof. Dr. H. Pichler), University of Vienna, Vienna, Austria.* During study of changes in periodontal membrane due to traumatic occlusion, several cases noted in which tissue between tooth and alveolar bone destroyed, and cartilage substituted for former connective tissue. Well known that cartilage forms on places where bone continually rubs against bone. When ends of bone fracture repeatedly move against one another, healing cannot occur—cartilage develops on ends, forming so-called false joint. Same process occurs in periodontal membrane, when tooth, by pressure against bone, destroys periodontal membrane and, through mastication, is continually forced against bone. *Fig. 9* shows apex of third molar. Owing to cusp interference, tooth pressed against alveolar bone, and necrosis of periodontal membrane occurred (*a*). Recurrent rubbing of tooth surface against alveolar bone developed cartilage-like tissue in periodontal membrane.

Fig. 10, higher magnification of cartilage-like tissue, shows homogeneous, partly fibrous, tissue with characteristic cartilage cells, some being calcified; some, uncalcified. (See *figs.* on p. 346.)

85. TUBERCULOSIS AND ACTINOMYCOSIS OF MANDIBLE; INFECTION THROUGH DECAYED TEETH. *Hans Pichler, M.D., Director, Dental Institute, University of Vienna, Vienna, Austria.* Teeth fill holes in epithelial covering of body like stopper closing bottle, presenting two avenues of diminished resistance, through which microörganisms often enter tissues: one, *through* tooth (periapical infections); another, *around* tooth (parodontal infections). Although most acute and chronic infections are caused by streptococci and other pus bacteria, Koch's bacillus and ray-fungus also often follow same paths. Following condensed histories of two cases present peculiar features:

H. R. (443), boy, 4, came to "Kieferstation" in 1920. Defect of mandible, left angle and part of ascending ramus. Nine months before, swelling began, resulting in loss of large piece of necrosed bone. Several teeth almost destroyed by decay; missing left deciduous molars probably decayed likewise. No sign of disease of lungs; submandibular lymphnodes very much swollen, especially on left side. Mother tuberculous (hemoptysis) when swelling around child's jaw began. History indicates bacilli from mother found way into body of child through pulp chamber of carious molar and produced *primary focus of tuberculosis* in mandible (two years later, 1922: tuberculosis in submandibular glands, microscopically found at biopsy). Dental splint reduced very marked dislocation of jaw; missing part regenerated in good position. Twelve years later (1934), recurrence: large tuberculous ulcer on gum and destruction of alveolar process around left incisors. Healed completely within six months after removal of involved teeth and extensive curetting of bone; existing tuberculosis of left apex of lung also healed. Boy now 19 and quite healthy.

K. L. (7079), girl of 11, came for treatment of ankylosis of right mandibular articulation and consequent underdevelopment of mandible; opening only 1 cm. between incisors. At 4, suffered from caries of bone in several fingers and toes; at 5.5, from swelling of right cheek and chin, with consecutive sloughing of several sequestra and establishment of sinuses in face and oral cavity. These presumably tuberculous affections of mandible and zygoma seemed healed completely; arthroplastic resection of joint undertaken to restore mobility and simultaneously to advance lower jaw and chin region of profile. Accomplished satisfactorily on right side; left side also necessarily lengthened mesiodistally—ascending ramus cut with wire saw; teeth then brought forward to good occlusion, and profile greatly improved. For retention, dental splint, constructed before operation, applied immediately afterward; worn until bony union on left side, and formation of good joint in improved position, completed.

Up to this point, nothing extraordinary in history; but patient did not make clear recovery. Post-operative swelling of cheek increased and, without rise in temperature, developed into abscess, which burst about three weeks after operation. To author's amazement, *actinomycosis fungus present in pus*. Inflammation and new sinus healed

readily under x-ray irradiation, and final result not impaired. Since contamination of wound with ray fungus during operation unlikely, fungus probably previously found way through decayed teeth or gingival pockets, slumbering somewhere within tissues until awakened to activity by operation. Or should it be supposed that primary infection of jaw bone had been not tubercular but actinomycotic, and had been slumbering there for more than five years? (Published in *Wiener klin. Wochschr.*, 1935, No. 20.)

86. RETENTION OF INLAYS AND ROOT-CANAL PINS. *Otto Preissecker, M.D., Dental Institute (Director Prof. Dr. H. Pichler), University of Vienna, Vienna, Austria.* Forces required to remove cast fillings, when fitting very well, badly, or very badly, determined. Similar studies made of pegs in root-canals—cast pegs, smooth and rough wires, screws and tubules. Found that fixity of inlays occurs only when cement applied in thin layer between tooth and inlay. Result explained by fact that cement has great resistance against *pressure*, but only slight against *pull*. Cement should therefore be applied in thin layer between metal and tooth, whether for inlays, root-pegs, or crowns.

87. ANESTHESIA WITH PLENOCAIN-SYMPATOL IN DIABETES. *Franz Schönbauer, M.D., Dental Institute (Director Prof. Dr. H. Pichler), University of Vienna, Vienna, Austria.* (No manuscript for publication.)

88. TRUE MIDDLE LINE OF JAWS IN ORTHODONTIC DIAGNOSIS. *A. Martin Schwarz, M.D., Vienna, Austria.* Former authors considered middle line between incisors, upper and lower, as real middle line also of jaws in anterior region. This opinion mistaken, for incisors like all other teeth *locally displaceable* within alveolar setting. In this displacement, alveolar bone can take part to such degree that *even papilla incisiva may deviate* from true middle line of upper jaw. Fremula labii and linguae may deviate with incisors, farther or nearer they are fixed to gingival margin. *One reliable point in anterior region of palate lies in raphe palatina on height of embranchment of second plica palatina transversa:* this is group of folds toward first bicuspids. This point is placed beyond alveolar bone; yet bone here, thick, is independent of natural irregularities of nasal cavities. Raphe palatina, in region of choanae, provides posterior "reliable point" on

middle line, for it is always symmetrically formed. Through these two points raphe-median-plane may be constructed. In symmetrical occlusion, admissible to transpose this plane to lower jaw also, and thus to find middle line of lower jaw; but in asymmetrical occlusion (for instance, Angle's Class II, subdivision), not permissible. In such cases true middle line of lower jaw must be found, for only such middle line helps to distinguish reliably local displacements of teeth from displacement of body of mandible itself. *Anterior point of true middle line of lower jaw given by spina mentalis* (*fig. 11, a*). Forms several prominences on lingual plane of chin-bone. There is—more precisely described—*pars superior* consisting of *couple of spines*, and *pars inferior* consisting of *only one spine exactly in middle of mandible*. Two spines of pars superior: insertions of musculus genioglossus; single spine of pars inferior, in exact middle: insertion of musculus geniohyoideus and of musculus mylohyoidea. By x-ray photograph of chin, from below, spina mentalis may be found—i.e., according to direction of main ray (*fig. 11*), two spines (*b*), or single spine (*c*), or three together (*d*). Besides, *remainders of cartilaginous symphysis* may be seen. Although halves of mandible grow together in first year of life, bony tissue often retains distinct traces of former sutura. Facial outlines of bony chin also often form distinct median prominence, *middle of protuberantia mentalis*, which with two lateral prominences are tubercula mentalia. By utilizing all details according to development, one can determine reliably whether and how far middle line of incisors deviates from real middle of lower jaw. Having thus found true middle line in anterior region of upper and lower jaws, halves of model put together so that true upper and lower middle lines, as found, cover each other in incisor region. By so doing asymmetrical occlusion changed into symmetrical one. Then, posterior point of raphe-median-plane may be transposed to lower jaw, when symmetroscope also shows exactly local displacements of teeth in lower jaw. (See *figs.* on p. 347.)

89. ABNORMAL RAMIFICATION OF LOWER ALVEOLAR NERVE. *Harry Sicher, M.D., Vienna, Austria.* In rare cases otherwise perfectly successful block anesthesia of lower alveolar (mandibular) nerve does not desensitize lower wisdom tooth. In such case author prepared mesio-occluso-distal cavities in both first and second right lower

molars, but unable to excavate small buccal cavity in third molar. After several such experiences and learning of others, this rare but typical occurrence found due to irregular ramification of lower alveolar nerve—high branching off of ramus dentalis posterior, i.e., small nerve normally innervating lower wisdom tooth. In abnormal cases, nerve leaves main trunk of mandibular nerve 10 to 15 mm. above mandibular foramen, and pierces bone through very small aperture on posterior circumference of basis of lingula mandibulae, i.e., close to anterior and superior circumference of mandibular foramen. Thence it courses downward and forward to apices of roots of lower wisdom tooth. By injecting anesthetic solution into lower portion of collar sulcus of mandible, main quantity lies on lateral-posterior surface of mandibular nerve. In abnormal cases, posterior ramus (i.e., nerve to lower wisdom tooth) escapes influence of anesthetic. Such failures can be corrected, and explanation experimentally confirmed, by injecting few drops of anesthetic solution several millimeters in front of place of normal injection.

90. NEW RESULTS OF BACTERIOLOGICAL INVESTIGATION OF PULPLESS TEETH. *Georg Stein, M.D., Jaw Department (Director Prof. Dr. H. Pichler), First Surgical Clinic (Director Prof. Dr. E. Ranzi), University of Vienna, Vienna, Austria.* Last year author reported data on "bacteriology of pulpless teeth" (*J. Den. Res.*, 1935–36, *15*, 199; June). One of most important results was finding that sterility at apices of non-treated dead teeth occurred in much larger proportion of cases than had been assumed. In continuation, author endeavored to learn whether there is difference between bacteriological flora at apex and in other parts of pulp canal. Apex cut off, contents of middle part of canal removed with sterile pulp-extractor, and sterile bur used to grind surface of canal to obtain dentin powder; each of three materials studied bacteriologically. For many teeth, no growth obtained with material from middle parts of pulp canal, although at apices different kinds of bacteria found. Sterility in only one instance at *apex* with growth from *middle* part of canal. Following conditions may explain this unexpected result: when one kind of bacterium grows in nutrient medium and no new nutrient added, individuals of that kind die out, although other kinds may grow for a time in that medium. Antivirus of Besredka prepared on this principle. In same

way, bacteria in middle parts of pulp canal, where no new nutrients arrive, may perish, while near apex nutrients enter through ramifications of pulp canal and there nourish bacteria. Author's observations seem to verify claim that, in treating infected teeth, therapeutic measures must extend to apex. At apices of infected teeth *various* kinds of bacteria often found. To determine which pathological, two series of experiments performed: (*a*) from all cultured strains, vaccines prepared and intracutaneous injections made in patients; (*b*) selected patients had acute periapical infections causing enlargement of regional lymph glands. Mouth, apices of teeth, and material from glands obtained by puncture, studied bacteriologically. Assumed that bacteria in glands were pathogenic. Vaccinations in first series of experiments gave negative results; nevertheless showed practically only *one* strain from one apex gave positive reaction. Second series showed that pure cultures of *hemolytic* streptococci usually obtained from inflamed lymph glands. Such streptococci also found at apices of teeth directly related to enlargement of lymph glands, but not at apices of other dead teeth in same mouth extracted at same time, nor in mouth flora and tonsils of same patient. Results confirm statement of Weber and Pesch that *hemolytic* streptococci often occur in acute periapical inflammations, but very rarely in chronic periapical processes. Show also that, of mixed flora at apex of infected tooth, only one kind of bacterium represented in enlarged regional lymph glands—condition indicating pathogenic strain. Since no hemolytic streptococci found in mouth flora, seems probable that nonhemolytic streptococci were transformed into hemolytic varieties.

91. TREATMENT OF PULP-INFECTED TEETH. *Richard Trauner, M.D., Vienna, Austria.* Author studied means (*a*) to clean root canal perfectly; (*b*) to disinfect by simple method; (*c*) to fill completely not only to apex, but closely against walls; and (*d*) to avoid or reduce irritations. Difficulties are due to narrowness of canal, and ensuing physical conditions. (*a*) *To clean canal*, after sufficient dilation, author introduces, nearly to apex, finest obtainable injection needle on right-angle syringe, and injects sterile water or dilute H_2O_2, driving out air and debris. To prevent injection of fluid beyond apex, and ensuing pain, canal must be made wide enough at all surfaces to permit fluid to flow easily beside needle to entrance of canal. Mate-

rial adhering to wall cannot be removed completely by syringing, but can be detached with Miller needle covered with cotton, and then flushed out by further syringing. (*b*) *For complete disinfection:* necessary completely to fill canal with non-irritating disinfectant and to retain long enough to kill microbes. Author prefers 7 percent chloramin solution, by injection technic described above; kills usual microbes in about 10 minutes; injected not only at two sittings, but immediately before filling. Patient waits for 15–20 minutes; and, as fluid cannot flow out of narrow canal, may stay in waiting room. (*c*) Having thus (*a-b*) perfectly cleaned and sterilized root canal, *must be filled completely* with material and in way to prevent entrance of liquid thereafter; so that chief canal can be eliminated as focus of infection. May be done with cement containing non-irritant disinfectant, chief difficulty being removal of air from canal, although residual bubbles may be unimportant if held in disinfected cement. From many experiments with very narrow glass-tubes, author concluded best to perforate apex so that air may escape, but not to dilate too much. Cement may be inserted best with fine probe, tip covered with amount so small that entrance of canal not obstructed by excess as tip is introduced. Probe must be pushed in and out frequently so that air may escape along sides coated with cement through entrance of canal. Finally, main channel of canal can be filled with hard point, which, if not quite stiff (guttapercha), may be compressed once gently in apical part and air-bubbles remaining there pushed away; but cannot be dissolved in cement as in chloropercha, and therefore operator should refrain from making breaks in cement, which cannot be filled again. (*d*) To get clear evidence on *ways to avoid irritations*, author statistically studied 400 of his cases. It is important not to dilate to apex at first sitting, and not to irritate periapical tissue with needles or chemically at sitting for filling. Author needs three sittings: (1) for dilation of three fourths of canal; (2) to complete dilation and to disinfect; (3) for filling immediately after final disinfection. If tooth shows signs of irritation, another sitting or watchful waiting, may be necessary. If tooth has fistula, can be filled at one or two sittings without fear of pain; just so, also, in non-swelling cases. With perfect disinfection and careful dilation, many irritations may be avoided. When teeth cannot be filled, author frequently uses

method of trepanation of bone to produce artificial fistula; performs trepanation subcutaneously in one minute, after localization of periapical abscess with U-shaped wire-loop as previously described (*J. Den. Res.*, 1935–36, *15*, 202; June).

92. RÔLE OF LEUCOCYTES IN SALIVA. *Josef Weinmann, M.D., Histological Laboratory (Director Prof. Dr. B. Gottlieb), Dental Institute (Director Prof. Dr. H. Pichler), University of Vienna, Vienna, Austria.* Most round cells in saliva are leucocytes ("salivary corpuscles") that steadily migrate through mucous membrane, chiefly gingivae. These cells give oxidase reaction and produce proteases. Average percentage amounts of proteolytic enzymes in caries-immune mouths much higher than in caries-susceptibles. Saliva of caries-immune individuals—especially sediment—kills bacteria that normally do not occur in saliva, or stops growth in 70 percent of all cases (shown with B. prodigiosus). This capability is weaker (50 percent) in saliva of caries-susceptibles; no difference, however, in counts of "salivary corpuscles." Equal amounts of leucocytes produce more proteolytic enzyme in mouths of persons immune to caries than of susceptibles. Immunity appears to be related to salivary leucocytes.

93. NEW METHOD OF MEASUREMENT FOR CONTROL OF ELECTRO-STERILIZATION OF ROOT CANALS. *Hermann Wolf, M.D., Dental Institute (Director Prof. Dr. H. Pichler), University of Vienna, Vienna, Austria.* According to rule of Faraday, quantity of substance separated during electrolysis is proportional to product of current and time. Measure of desired bactericidal effects during electro-sterilization is coulomb value. In dentistry, "m.a.min.," sixty times more than coulomb, is unit. Electro-sterilization, for bactericidal effects in root canals and surrounding tissues, regarded as complete when patient receives 30 m.a.min. In practice, difficulties in measuring m.a.min. arise from current fluctuations during treatment. To overcome difficulty, apparatus, "Sternax-controller" (*fig. 12*), constructed to measure, during electro-sterilization, both coulombs used and m.a.min. applied, combining automatically units of current and time, simplifying electro-sterilization and making it more exact. Instrument can be connected with old apparatus for electro-sterilization, or with new apparatus combined with instrument. (See *fig.* on p. 348.)

B. CONTRIBUTIONS BY OTHER MEMBERS OF THE ASSOCIATION, AND BY GUESTS, WHO COULD NOT BE PRESENT: ABSTRACTS 94–120

94. FILAMENTOUS BACTERIA OF MOUTH. *Basil G. Bibby, B.D.S., Ph.D., Senior Rockefeller Fellow in Dentistry, School of Medicine and Dentistry, University of Rochester, Rochester, N. Y.* Detailed analysis of confused literature on nomenclature and properties of filamentous oral bacteria. Reported organisms classified in accepted genera. Those showing definitely filamentous form probably belong to genus *Leptotrichia*. Five groups of unbranched bacterial filaments distinguished by microscopic examination. Cultivated strains of bacterial filaments separated into seven groups on basis of morphology, staining reactions, and growth characteristics; only two definitely resemble previously described species. Organisms of genus *Leptotrichia* have closer kinship to genera *Fusiformis* and *Corynebacterium* than to genus *Actinomyces*. Suggested that genus *Leptotrichia* be transferred from family Actinomycetaceae to family Mycobacteraceae. Possible significance of *Leptotrichia* in causation of caries and salivary calculus discussed. (Thesis: submitted to University of Rochester in partial fulfillment of requirements for Ph.D. degree.)

95. pH OF NORMAL RESTING SALIVA. IV: RELATIONSHIP TO DENTAL CARIES. *Robert E. Brawley, B.S., M.S., D.D.S., School of Medicine and Dentistry, University of Rochester, Rochester, N. Y.* Colorimetric pH determinations (Clark and Lub) on resting saliva of 281 school children; male and female, average age 13.2 years. Data correlated with quantitative caries-indices (method of Mellanby, modified by Day and Sedwick). pH indices established at beginning and end of 12-month period. No relationship existed at either examination, but when increase in caries index compared with pH of salivas, definite relationship expressed itself as (*a*) low pH ranges, high caries indices; (*b*) high pH ranges, low caries indices.

96. BACTERICIDAL ACTION OF SALIVA. II: FACTORS THAT INFLUENCE INHIBITORY AGENT. *Robert E. Brawley, B.S., M.S., D.D.S., and H. Jobe Sedwick, D.D.S., School of Medicine and Dentistry, University of Rochester, Rochester, N. Y.* Various media and species of organisms, and resting and activated samples of saliva from male and female subjects, used. Following factors influenced inhibitory

agent, positively or negatively: (*a*) *Saliva*—incubation, refrigeration, filtration, centrifugation, dilution, heat, cold, acids, alkalies, pH, rays, and amount per "well." (*b*) *Saliva and medium*—incubation and refrigeration. (*c*) *Subject*—salivary Ca, P, Mg, mucin, corpuscles, rate of flow, pH, viscosity, diurnal and day-to-day variations. (*d*) *Medium*—kind, amount per plate, depth of "well," diameter of "well," pH, and refrigeration. (*e*) *Organism*—strain used, and amount of inoculum.

97. FURTHER INFORMATION ON DISTRIBUTION OF CHRONIC ENDEMIC DENTAL FLUOROSIS (MOTTLED ENAMEL) IN UNITED STATES. *H. Trendley Dean, D.D.S., National Institute of Health, U. S. Public Health Service, Washington, D. C.* Endemic dental hypoplasia (mottled enamel) associated with use, during development of permanent teeth, of drinking water containing toxic amounts of fluorides. Minimal threshold not yet established, but less than 1 part per million of flourine (F) no public-health significance from dental standpoint. Water-borne disease, problem particularly important in preventive medicine. Careful surveys show quantitative relation between concentration and clinical effect, regarding both incidence and percentage distribution of severity, particularly latter. Other possible signs or symptoms of fluorosis not determined. Since 1933 marked increase in number of known endemic areas (*table 2*). Most severely affected state is Texas. Fact that large cities—Amarillo, Lubbock, and Plainview—are located in region of greatest severity makes this serious problem of keen public-health interest. West Texas region about equal in size to Pennsylvania. Of 325 endemic areas in U. S., 89 (about 27 percent) in Texas. Number of persons affected: 84 endemic communities each having population of 1000 or more distributed among sixteen states—45 (about 54 percent) in Texas.

98. CRYSTALLOGRAPHIC STUDIES OF DENTAL ENAMEL. *Moses J. Eisenberg, D.M.D., F.R.M.S., Dental School, Harvard University, Boston, Mass.* Mineralogical methods applied to fragments of enamel; crystal system and many other optical constants obtained, such as indices of refraction, extinction angles, study of interference colors, sign of elongation, and optic sign. Histological details and interpretations enhanced and elucidated by application of optical methods of study.

99. BACTERIOLOGIC EXAMINATIONS OF STOCK PROCAIN SOLUTIONS. *L. I. Grossman, D.D.S., D.M.D., Philadelphia, Pa., and J. L. T.*

TABLE 2

Number of mottled-enamel areas in United States; by states

STATE	NUMBER OF AREAS EITHER (A) REFERRED TO IN LITERATURE AS DEFINITELY ENDEMIC; OR (B) ENDEMICITY CONFIRMED BY U. S. P. H. S. SURVEYS		NUMBER OF RELIABLY REPORTED ENDEMIC AREAS, NO CONFIRMATORY SURVEYS		TOTAL NUMBER OF MOTTLED ENAMEL AREAS SURVEYED, PLUS "REPORTED" AREAS	
	1933[1]	1936	1933[1]	1936	1933[1]	1936
Arizona	45	49	1	1	46	50
Arkansas	1	0[2]	2	2	3	2
California	5	5	1	2	6	7
Colorado	11	34	1	0	12	34
Idaho	2	1	1	2	3	3
Illinois	2	4	4	5	6	9
Indiana	0	1	0	0	0	1
Iowa	1	13	0	0	1	13
Kansas	4	5	0	0	4	5
Louisiana	0	0	0	1	0	1
Minnesota	1	1	1	1	2	2
Mississippi	0	1	3	1	3	2
Nevada	0	5	8	4	8	9
New Mexico	1	1	13	16	14	17
North Carolina	1	2	1	7	2	9
North Dakota	1	2	6	7	7	9
Ohio	0	2	0	0	0	2
Oklahoma	0	0	4	4	4	4
Oregon	0	0	2	2	2	2
South Carolina	4	9	0	1	4	10
South Dakota	12	13	16	19	28	32
Tennessee	2	2	0	0	2	2
Texas	2	75	25	14	27	89
Utah	0	0	3	3	3	3
Virginia	2	2	6	6	8	8
	97	227	98	98	195	325

[1] Pub. Health Rep., *48*, 25; June 23, 1933.

[2] Communal water-supply changed in 1928; local reports indicate that hypoplasia is not developing among children born since that date.

Appleton, B.S., D.D.S., Sc.D., School of Dentistry, University of Pennsylvania. Fifty stock procain solutions, available on market, examined culturally and by injection into white mice for presence of

microörganisms. Media used designed to reveal ordinary pyogenic cocci, streptococci, anerobes, yeasts, molds. Mice used to reveal *B. anthracis*, *Cl. tetani*, and bacteria of gas gangrene. Only isolated microörganisms of possibly pathogenic significance were yeasts, found culturally in three preparations. Detailed report to be submitted to Council on Dental Therapeutics.

100. pH OF STOCK PROCAIN SOLUTIONS. *Louis I. Grossman, D.D.S., D.M.D., Philadelphia, Pa.* Procain solutions, in all-glass ampules, rubber-stoppered ampules (carpules, anestubes, novampuls), and rubber-stoppered bottles, tested for pH. Age of solutions varied from 4 to 16 months, condition favoring manufacturer from standpoint of pH, since solutions on aging become more alkaline owing to slow solution of free alkali from glass container, but aging tends to impair anesthetic effectiveness. pH determined by Youden potentiometric apparatus, using Cullen quinhydrone electrode and saturated calomel cell. Accuracy checked, from time to time, with standard buffer-solution of known pH. Tested 53 batches of solution, from original containers, representing products of seven manufacturers. *Conclusions:* (*a*) Stock procain solutions from same batch vary greatly in pH. (*b*) pH of almost all solutions far below that of human tissue-fluids. (*c*) Products of three manufacturers averaged slightly above pH 6. (*d*) Products of four manufacturers averaged below pH 5. (*e*) pH of stock procain solutions is variable and unreliable factor.

101. PROBABLE PERCENTAGE OF CORRECT GUESSES REGARDING STERILITY OF ROOT CANALS WITHOUT BACTERIOLOGICAL CONTROL. *Louis I. Grossman, D.D.S., D.M.D., Philadelphia, Pa.* Most dentists probably do not use bacteriological means to determine sterility of root canals before filling. Instead, appearance and odor of root-canal dressing are criteria for filling. Present study intended to determine how often bacteriological status coincided with clinical appearances. Coöperation of several dentists, who do not ordinarily use bacteriological controls, enlisted. When coöperating dentist felt canal was ready for filling, judged by appearance and odor of dressing, culture in hormone broth examined after 48-hour incubation at 37°C. Cultures taken from root canals of 150 teeth. By this culture method to evaluate percentage of correct guesses regarding sterility of root canals, when judged by clinical criteria alone, average of correct guesses was 58 percent. Percentage higher, when smears made as

preliminary to culture taking. With only slightly better than even chance for guessing correctly status of root canal, *great need for bacteriological control apparent.*

102. NEW AND SCIENTIFIC METHOD OF PRODUCING TEMPOROMANDIBULAR-ARTICULATION RADIOGRAMS. *L. B. Higley, B.A., M.S., D.D.S., Dental School, State University of Iowa, Iowa City, Iowa.* Author described new method of approach in radiographing temporomandibular joint; methods of investigation employed to study angulations of joint with respect to head; and machine used to orient head to reproduce angles for correct radiographic procedure.

103. STUDY OF DENTAL CALCIFICATION BY MEANS OF X-RAY ABSORPTION COEFFICIENTS. *Franklin Hollander, Ph.D., Dental School, Columbia University, New York City.* Teeth are frequently said to undergo normal post-eruptive changes in degree of calcification. Such changes must be studied spatially, as *density of calcification;* i.e., in terms of weight of Ca and PO_4 *per unit volume,* rather than *per unit weight,* of sample. Latter gives erroneous measure of spatial distribution; e.g., 10 percent difference in mgm. Ca/mm.^{3} of enamel corresponds to 1.3 percent in terms of mgm./100 mgm. However, weight of Ca or PO_4 per mm.^{3} cannot be determined with satisfactory precision, but linear x-ray absorption coefficient, being exact measure of radiopacity, yields very satisfactory criterion of density of calcification. To study problem, method devised for determining absorption coefficient: (*a*) applicable to 1/4 mm.^{3} of tooth substance; (*b*) permits correlation with histological structure; (*c*) has P.E. = $\pm$ 0.3 percent; (*d*) gives mean results in satisfactory agreement with calculated values. Mean ratio of coefficients for human enamel and dentin (En/D): 1.9 (observed) against 1.8 (calculated from typical analysis), excellent agreement with ratio calculated in terms of mgm. Ca/mm.^{3} (1.8); differs from that in terms of mgm. Ca/100 mgm. (1.3). Mean coefficient in coronal dentin of four human teeth (ages 17–68) constant to $\pm$ 1 percent. Individual values decrease as position of measurement passes into roots, indicating gradient in calcification parallel with direction of dentin formation. (Summary of data in preliminary reports: *Proc. Soc. Exp. Biol. Med.*, 1935, *33*, 388; *Ibid.*, 1936, *34*, 158; *Proc. Am. Soc. Biol. Chem.*, 1936, *30*, lii. Aided by grant from Commonwealth Fund of N. Y.)

104. THERMAL CHANGES IN ENAMEL, DENTIN, AND PULP CHAMBER

INDUCED BY VARIOUS OPERATIVE PROCEDURES. *Paul H. Jeserich, A.B., D.D.S., Dental School, University of Michigan, Ann Arbor, Mich.* (No manuscript for publication.)

105. FACTORS IN SALIVA CORRELATED WITH PRESENCE AND ACTIVITY OF DENTAL CARIES. *Maxwell Karshan, M.A., Ph.D., Medical and Dental Schools, Columbia University, New York City.* Determinations made for CO_2-capacity, NH_3 nitrogen, total Ca, inorganic PO_4, and protein; and fractions of Ca, PO_4, and protein removed from saliva shaken with tricalcium phosphate. Saliva, stimulated by chewing paraffin, collected in two portions, approximately 15 minutes apart, 1–3 hours after breakfast. First portion (15 cc.) used for determination of Ca, PO_4, and protein; second (8 cc.), for CO_2-capacity and NH_3 nitrogen. Tricalcium phosphate (Mallinckrodt), heated for at least 24 hours at 100°C., used to remove fractions of Ca, PO_4, and protein—5 grams added to 10 cc. of saliva; mixture, in glass-stoppered bottle, shaken mechanically for two hours, centrifuged, and supernatant fluid analyzed. Stimulated saliva of caries-free and caries-arrested groups differed significantly from that of caries-active group: (1) caries-free and caries-arrested groups had higher values for CO_2-capacity; (2) higher values for percent of Ca removed; (3) lower values for percent of PO_4 removed. Mean values for total Ca and inorganic PO_4 in caries-free and caries-active groups indicate differences similar in magnitude and direction to those previously reported (Karshan, Krasnow, and Krejci: *J. Den. Res.*, 1931, *11*, 573). Similar differences in mean values, in these respects, found between arrested-caries and caries-active groups. These differences may assume significance, if larger groups give similar results. Values for protein and NH_3 nitrogen showed no significant differences. (Aided by grant from Carnegie Corporation. (To be published in *J. Den. Res.*)

106. ULTRAFILTRATION AND ADSORPTION STUDIES: SIGNIFICANCE OF SALIVARY CALCIUM AND INORGANIC-PHOSPHORUS PARTITION IN RELATION TO DENTAL CARIES. *Frances Krasnow, A.M., Ph.D., and Edith B. Oblatt, B.A., Guggenheim Dental Clinic, New York City.* Previous investigation on fractionation of salivary Ca and P, by ultrafiltration and adsorption, indicated difference in condition of Ca in salivas of caries-free and caries-susceptible individuals. Thus, in 67 percent of

former group, less than 50 percent of Ca was ultrafiltrated; in 65 percent of latter, more than 50 percent. Difference suggests stable combination of Ca with colloidal component. Stable salivary Ca-proteinate previously suggested, for caries-free persons, on finding more protein remained in supernatant fluid after centrifugation of saliva than before. Present results seem to support this probability. Significant differences in P not found. Adsorption partition tends to depend on adsorbent employed. One specimen of $BaSO_4$ adsorbed 90 percent of salivary Ca and 56 percent of P; another, 65 percent of Ca and 97 percent of P. Sample of $Ca_3(PO_4)_2$ removed 89 percent of Ca; P indeterminate. Representative $BaSO_4$—composite of twenty-six individual precipitations obtained by mixing $Ba(NO_3)_2$ and H_2SO_4 under identical conditions—showed tendency to adsorb more ultrafiltrable Ca for caries-free series than for caries-susceptible.

107. COLLECTION OF HUMAN SALIVA FOR QUANTITATIVE BIOCHEMICAL STUDIES. *Frances Krasnow, A.M., Ph.D., Edith B. Oblatt, B.A., and Naomi Kaplan, B.A., Guggenheim Dental Clinic, New York City.* Following conditions should be met: (*A*) *Preparation of patient before collection*—(*a*) Definite physiological plane for at least one week preceding analysis, to preclude unusual or cyclic dento-medical symptoms, unless included in projected investigation. (*b*) Detailed record of diet for week preceding analysis. (*c*) Eight hours of sleep night before each test-day. (*d*) No brushing of teeth or rinsing of mouth, nor breakfast or smoking, on morning of test day. (*e*) Interval of 1.5 to 2 hours between rising and collection, to include 15-minute rest at laboratory. (*B*) *Collection for analysis*—(*f*) Saliva for pH, CO_2, or other labile components: minimum volumes obtained by expectoration of spontaneous flow. (*g*) For Ca, Mg, and similar positive ions—and for PO_4 (recorded as P), and similar negative ions, protein, and lipids—total of approximately 40 cc. of spontaneous secretion collected with aid of suction and dental ejector. Mixed secretion obtained, and time taken to secure it recorded. Composition of samples (*f-g*) approximates very closely that of similar volume of mixed saliva in spontaneous flow. To obtain comparatively large samples from very young children, suction employed. Paraffin-stimulated post-absorptive saliva showed apparently same reliability in concentration and duplication for protein, Mg, and perhaps Ca, but yielded much lower

P figures. Stimulated salivation is precluded in study of salivary composition in infants—field now receiving increasing attention.

108. SALIVARY LIPID PHOSPHORUS: ALCOHOL-ETHER SOLUBLE PHOSPHORUS. *Frances Krasnow, A.M., Ph.D., and A. S. Rosen, D. Phar., Guggenheim Dental Clinic, and New York Post-Graduate Medical School and Hospital, New York City.* Analyses of salivary lipid-P for 80 cases indicated (*a*) technic employed (*J. Lab. Clin. Med.*, 1935, *20*, 1090) yielded accurately reproducible results. (*b*) Concentration ranged from 0.05 to 1.79 mgm. per 100 cc. of saliva—55 percent below 0.2 mgm. (*c*) In dento-medical clinically-normal group, low values (0.05–0.20, average 0.16 mgm., percent) appeared characteristic for younger individuals (6 months–20 years); higher figures (0.19–0.48, average 0.29 mgm., percent) for persons above 20. (*d*) Caries-susceptible subjects at corresponding age-levels showed salivary-lipid-P content within respective limits: 0.09–0.38, average 0.22 mgm., percent, and 0.13–0.50, average 0.27 mgm., percent. (*e*) Concentrations above 0.50 mgm. obtained in cases having miscellaneous dental disorders. (*f*) Lipid-P tends to higher values in individuals having tooth abnormalities, thus simulating trend for cholesterol, although not to same extent.

109. ODONTOBLASTS AND DENTIN FORMATION. *M. W. McCrea, D.D.S., Rockefeller Fellow in Dentistry, School of Medicine and Dentistry, University of Rochester, Rochester, N. Y.* Cytology of odontoblasts in various stages of tooth development; particular attention given to reticular apparatus of Golgi, on earliest stages up to and including five-day-old rats. *In earliest stages*—no dentin present—cells then differentiating into odontoblasts are small, and rather poor in material capable of staining with osmic acid; techniques employed were Kolatchev method, as modified by Nassonow, and $AgNO_3$ method of Da Fano. *As odontoblasts develop*, osmiphilic particles hypertrophy and assemble in cell pole nearest dentin. Using reticular apparatus as index to cellular activity, formation of dentin appears to be dependent on, or associated with, increased odontoblastic activity. Further work, throughout entire developmental period, projected.

110. PROGRESS OF NATION-WIDE SURVEY OF CHILDREN TO DETERMINE DENTAL NEEDS. *C. T. Messner, D.D.S., F.A.C.D., U. S. Public*

Health Service, Washington, D. C. The nation-wide survey to determine dental needs and past treatment for children of school age has been completed. This survey covers a cross section of twenty-six states and includes 1,600,000 dental examinations. All examinations were made by dentists using a mouth mirror and explorer. More than 12,000 dentists participated in the examinations, which were made of approximately 40 percent of the children of school age in Minnesota, Indiana, New Jersey, and Tennessee. In the other twenty-two states, small cross-sections were examined. The results will be shown in more than 1,700 areas in the twenty-six states, and have been tabulated according to size of city or town and rural area. The *Bulletin*, showing tabulated results of this survey and comprising 398 pages, will be available for purchase from the Government Printing Office about May 30, 1936. The cost should not be more than 50 to 75 cents each. In ordering the *Bulletin*, write to the Government Printing Office, Washington, D. C., and ask for *Public Health Bulletin No. 226*. [Published as submitted on May 26, 1936, by Dr. Messner, whose untimely death occurred two days later.—*Ed.*]

111. RELATION OF SERUM INORGANIC-P TO MACROSCOPIC DENTAL CARIES IN ALBINO RATS. *Isaac Newirth, Ph.D., College of Dentistry, New York University, New York City.* Author reported confirmation of findings of Hoppert, Webber, and Canniff on production of macroscopic dental caries in rats by inclusion of coarse rice or corn in diet. Oatmeal ground in Wiley mill, using same sieve that produces coarse corn or rice, or corn or rice sifted through 60-mesh sieve, ineffective in this respect. Assumption by Klein and McCollum (*Science*, 1931, *74*, 662), that Hoppert's results due to different contents of Ca and P in diet, rather than to particle size, while logical, controverted by results obtained with diets of same chemical composition but of different physical consistency as regards particle size. Author carried out such experiments, using rice as cereal. With coarse rice, macroscopic caries produced; with fine rice, no such caries. Similar observations reported by Klein and McCollum, using cornmeal in rachitic diet, and by Rosebury and coworkers. In Klein-McCollum paper referred to above, suggestion made—based on work of Kramer and Howland on serum inorganic-P on varying P levels in diet, and on dietary P in Hoppert's work—that critical level of serum inorganic-P

existed in relation to dental caries in rat. This level, by calculation, was postulated by Klein and McCollum to be 10.5 mg. (±0.5) of P per 100 cc. of serum: below this level, rats develop caries; above this level, immune to caries. In author's feeding experiments with Hoppert's rice diet of same chemical composition, but of different particle size: no serum difference in inorganic-P content between two groups. Diet containing coarse rice produces caries; one containing fine rice, no caries. [Preliminary reports: meetings of (*a*) N. J. State Dental Society, April 13, 1932; (*b*) N. Y. Section, I. A. D. R., May 25, 1933.]

112. NUTRITION AND DENTAL CARIES AMONG EASTERN AND CENTRAL AFRICAN TRIBES. *Weston A. Price, D.D.S., M.S., F.A.C.D., Dental Research Laboratories, Cleveland, Ohio.* One of greatest difficulties in studies of etiologic factors in loss of immunity to dental caries has been absence of adequate control groups having very high immunity. To find and study such groups, author examined isolated remnants of primitive tribes, including groups in Switzerland, Outer Hebrides, Alaska, northern and central Canada, United States, and South Sea Islands; in 1935 studied several native tribes in eastern and central Africa. Of 27 native tribes, all of different tribal stock, all individuals examined in 5 were free from caries; these groups contained 84 individuals with 2370 teeth. In 13 of 27 groups, immunity to caries was so high that only 13 individuals in 328 (3.9 percent) had evidence of caries past or present; of 9564 teeth, only 26 (0.2 percent) carious. In contrast: of 8 other tribal groups containing 577 individuals with 16,514 teeth, 46.8 percent of all individuals had caries; 6.8 percent of all teeth involved. These 8 groups lived in considerable contact with foreigners, and used modernized menus higher in cereals and sweets, and lower in animal products, than isolated groups having high immunity. (To be published in *J. Amer. Den. Assoc.*)

113. NUTRITION AND DENTAL-ARCH DEFORMITIES AMONG EASTERN AND CENTRAL AFRICAN TRIBES. *Weston A. Price, D.D.S., M.S., F.A.C.D., Dental Research Laboratories, Cleveland, Ohio.* Irregularities of dental arches have been ascribed chiefly to postnatal influences, with much emphasis on thumb sucking, faulty breathing habits, and premature loss of deciduous teeth; very elaborate procedures developed for correction of dental-arch deformities by mechanical means. Exceedingly little material in literature throws light upon prevention

or rôle of nutrition in etiology of facial and dental-arch deformities, excepting that of author's field studies among primitive racial stocks, continued, in 1935, in eastern and central Africa. In critical study of 242 persons, in seven relatively isolated primitive tribes, only 10 (4.1 percent) had irregularities of dental arches or of individual teeth. Of 92 natives, born in five modernizing missions, 45 (48.8 percent) had dental-arch deformities. Individuals in former group, throughout both formative and growth periods, were under influence of primitive nutrition. Formative and early growth periods of individuals of latter group occurred under influence of modernized dietaries. Several isolated native tribes placed much stress not only on food of expectant mother and growing child, but also on food used before and at time of conception.

114. CHEMICAL EXPRESSION OF SALIVA RELATED TO IMMUNITY TO DENTAL CARIES. *Weston A. Price, D.D.S., M.S., F.A.C.D., Dental Research Laboratories, Cleveland, Ohio.* Previous communications presented data relating chemical factors of saliva to level of individual immunity to caries—two factors shown to be directly related to nutrition (*Den. Cosmos*, Dec. 1932; *J. Amer. Den. Assoc.*, Aug. 1933; March 1936). Detailed procedures for chemical analysis of saliva in press; in general, relates to behavior of inorganic P when saliva shaken with finely powdered bone. In high immunity to caries, marked lowering of reading for inorganic P for saliva shaken with powdered bone, as compared with reading without powdered bone; whereas, in cases of lost immunity to caries, marked increase in reading for inorganic P, or at least lessened decrease. This procedure in use, in clinical practice for five years, now provides data from 2429 chemical analyses in relation to caries as determined clinically with x-rays. Reinforcement of nutrition, with both fat-soluble activators and natural foods supplying minerals liberally—particularly P—caused, in 121 individuals having caries, salivary increase in inorganic P, averaging 8.9 percent. After this reinforcement, inorganic P in saliva shaken with powdered bone decreased (when compared with sample without powdered bone) 10.8 percent—total change of 19.7 percent. Coincident with improvement in chemical reaction of saliva, on above basis, roentgenograms revealed increased density of tooth structure associated with cessation of active caries

115. SIGNIFICANCE OF "CIRCULATORY CANALS" IN ROENTGENOGRAMS OF ALVEOLAR BONE. *William B. Ryder, D.D.S., George Williams Hooper Foundation for Medical Research, and Dental School, University of California, San Francisco, Calif.* (No manuscript for publication.)

116. BACTERICIDAL ACTION OF SALIVA. I: SELECTION OF MEDIA FAVORABLE TO GROWTH OF CERTAIN ORGANISMS, TO DEMONSTRATE INHIBITION BY SALIVARY AGENT. *H. Jobe Sedwick, D.D.S., and Robert E. Brawley, B.S., M.S., D.D.S., School of Medicine and Dentistry, University of Rochester, Rochester, N. Y.* Attempt to find solid media suitable for growth of certain species of bacteria; also to facilitate demonstration of bactericidal action of saliva, using Fleming's "well" method. Species and media finally selected as most satisfactory: (*a*) *Lactobacillus acidophilus* (Hadley's Type I); galactose whey, 1 percent agar plus horse serum, pH 6.4. (*b*) *Staphylococcus pyogenes aureus* (isolated at Strong Memorial Hospital); Kulp's tomato juice, 1 percent agar, pH 6.3. (*c*) *Streptococcus hemolyticus, Beta* (isolated at Strong Memorial Hospital); Savita, 1 percent agar plus horse serum, 1 percent. (*d*) *Micrococcus lysodeikticus* (Dr. Fleming's); plain agar 2 percent, pH 7.4.

117. MECHANICS OF MANDIBULAR SLING. *M. Russell Stein, D.D.S., Dental School, Columbia University, New York City.* Masseter and internal pterygoid are companion muscles. Both pull jaw upward and forward. Masseter is outside of ramus; internal pterygoid, inside. Suspension of angle of jaw by these two muscles is like action of a sling. Mandible is so formed as to best withstand external compressive forces brought to bear upon it by muscles of sling.

118. EFFECTS OF PARTICLE SIZE UPON PHYSICAL PROPERTIES OF SILICATE CEMENTS. *Edgar W. Swanson, D.D.S., M.S.D., Northwestern University Dental School, Chicago, Ill.* Commercial brand of silicate-cement powder separated into three different grades as to particle size, hereafter designated *large, medium,* and *small.* Cements made from each compared with cement from original powder, as to consistency, setting time, crushing strength, penetration, shrinkage, translucency, solubility, under exact conditions of temperature, humidity, powder:liquid ratio. Slab temperature, and humidity during mixing, 70°F. and 65 percent, respectively. All specimens

allowed to set in atmosphere of 100 percent relative humidity and 37°C. Results (*table 3*) indicate powders composed of *uniformly fine* particles made most satisfactory cements. (Thesis: submitted in partial fulfillment of requirements of Northwestern University for degree of Master of Science in Dentistry. (Published in *J. Amer. Den. Assoc.*, 1936, *23*, 1620.)

119. ORAL ASPECTS OF AMYOTROPHIC LATERAL SCLEROSIS. *Bertram L. Wolfsohn, D.D.S., University of California and San Francisco Polyclinic and Post-Graduate School, San Francisco, Calif.* This disease of central nervous system, having uncertain etiology, manifests

TABLE 3

Effects of particle size upon physical properties of cements

CONDITIONS	POWDER			
	Original	Large	Medium	Fine
Size	>1–60μ	12–60μ	4–12μ	Under 4μ
Relative distribution		16%	34%	50%
Setting time	3′38″	4′5″	3′5″	2′5″ 2′45″[1]
Crushing strength (lbs./in.²)	8,558	4,790	10,327	9,604
Penetration	Slight	Considerable	Slight plus	Practically none
Shrinkage	Slight	Considerable	Slight plus	Practically none
Translucency	Greatest	Slightly less	Less	Least
Solubility	...	...	...	Least

[1] Using 1.1 gm. of powder to 0.4 cc. of liquid—ratio used for all succeeding data; 1.3 gm. of powder to 0.4 cc. of liquid used for preceding data.

itself by progressive muscular atrophy of extremities. Oral symptoms, occurring as disease attacks bulbar nucleii of central nervous system, include glossal fibrillation and atrophy, salivation, dysphagia and dysarthria, labials being particularly indistinct. Oral treatment merely palliative. No extensive restorations should be attempted, but scrupulous attention given to establishment and continued maintenance of mouth hygiene; Viogen especially useful.

120. ALLEGED RELATION OF SALIVARY AMMONIA TO DENTAL CARIES. *Guy E. Youngburg, M.S., Ph.G., Ph.D., Medical School, University of Buffalo, Buffalo, N. Y.* Author determined variations in amounts of salivary NH_3 for normal individuals (non-carious) at different times

of day, on different days, and under various conditions. Values ranging from 1.28 mgm. to 13.66 of NH_3-nitrogen per 100 cc. of saliva, under normal conditions, obtained—one value, 16.35 mgm., immediately after smoking; average, approximately 8.20 mgm. Salivary NH_3-nitrogen values for 45 individuals having active dental caries ranged from 3.57 mgm. to 16.96 per 100 cc.; average, 8.23—practically same as for non-carious individuals. Claim of Grove and Grove, that low concentration of NH_3 in saliva not exceeding 4 mgm. of NH_3-nitrogen per 100 cc., is main factor causing susceptibility to dental caries, not substantiated. (Published in *J. Den. Res.*; this issue, p. 247.)

XII. Necrology[4]

WILHELM DIECK: 1867–1935

Wilhelm Dieck died very suddenly on Feb. 28, 1935. He was born on Jan. 12, 1867, in Haensel, near Essen in Westphalia, son of a mining official. After passing his final examination at the gymnasium in 1885, he studied dentistry at Berlin University. In 1887 he passed state-board examinations in Berlin with honors. He continued at Berlin University, first as a postgraduate student with Prof. Dr. Busch, later as an assistant. He collaborated a long time in Prof. Dr. Miller's scientific investigations, and received the M.D. degree in 1896 at Würzburg. In 1899 he became Assistant Professor in the operative department; in 1907, "ausserordentlicher Professor" and head of the department, succeeding Prof. Dr. Miller; in 1924, Dean of the College of Dentistry, University of Berlin, which position he held until his death.

It is perhaps for Dr. Dieck's important work in x-ray photography that he is best known. Very important also was the success of his rehabilitation of the amalgam filling. Many publications indicate the extent of his research. Perhaps one of the best known is *Anatomy and Pathology of the Teeth and Jaws in X-ray Photography* (1911). He was a leader in dental societies, and held a number of important positions on various committees and councils. For many years he

[4] First annual report of the Necrology Committee, Frederick B. Noyes, Chairman. The Committee was appointed at the general meeting in 1935 (*J. Den. Res.*, 1935–36, *15*, 226).

was President of the Society of German University Teachers in Dentistry. In 1915 he received the honorary degree of Doctor of Science at the University of Pennsylvania. In 1930 he gave a series of lectures in Japan at the request of the University of Japan. He became very influential in the dental profession in Europe. He was a splendid representative of German science and thoroughness, and his loss will be keenly felt by the dental profession in all nations.

CHALMERS J. LYONS: 1874–1935

Chalmers J. Lyons died on May 18, 1935. He was born in Martinsburg, Ohio, on April 30, 1874, son of John P. and Manilla (White) Lyons. He received his early education at the Central Michigan Normal School, Mt. Pleasant, Mich. From there he went to the University of Michigan, where he received the D.D.S. degree in 1898 and the D.D.Sc. degree in 1911. He practised in Adrian, Mich., until 1907, when he moved to Jackson, Mich., where he was associated in practice with Dr. J. W. Lyons. In 1909 he married Grace B. Driggs, of Palmyra, Mich. They had one son: Richard Hugh. After moving to Ann Arbor, Mich., he became, in 1907, an instructor in clinical dentistry at the University of Michigan; in 1913, Instructor in Oral Surgery and Consulting Dentist to the University Hospital; in 1915, Prof. of Oral Surgery and Consulting Dental Surgeon; in 1934, Chairman of the Executive Committee of the School of Dentistry. He was a member of the American Association of Oral and Plastic Surgeons (President from 1933 to 1934), American Dental Association, American Medical Association, American College of Dentists, Michigan State Advisory Council of Health, Sigma Xi, Phi Kappa Phi, Omicron Kappa Upsilon, and Delta Sigma Delta; also a past president of the Michigan State Dental Society.

During the years of his service, Dr. Lyons contributed largely to dental and medical literature in the field of oral surgery. He was the author of a text entitled *Fractures and Dislocations of the Jaws*. He also contributed to the *American Textbook of Operative Dentistry* (Ward), and to *Oral Surgery* (Mead). Dr. Lyons received many honors and awards in recognition of his distinguished service. In 1933 he received the Jarvie Medal from the New York State Dental Society. He was friendly and kind. While we deeply mourn his

passing, we are profoundly grateful for his splendid life. His influence will now take even deeper roots in the lives of those who knew him; and, through them, an unspoken wealth will broaden with the passing years.

ARTHUR T. ROWE: 1883–1935

Arthur Taylor Rowe died on Dec. 12, 1935. He was born in Casselton, N. Dak., on May 12, 1883, son of Dr. H. J. and Helen (Taylor) Rowe. His father was a prominent physician of that community. His early education was obtained at the Casselton High School, later at the University of Minnesota, where he graduated from the School of Dentistry in 1906. In 1908 he married Marion Degman of Winona, Minn. He began dental practice in Larimore, N. Dak. In a few years he moved to Minneapolis. In 1924 he became an instructor in dental prosthetics at the University of Minnesota, where he remained as Professor of Prosthetics until 1926, when he became Professor of Prosthetics and head of the division of prosthetic dentistry at the School of Dental and Oral Surgery, Columbia University, New York. In 1932, he was appointed Associate Dean. Dr. Rowe was actively associated with the dental societies of Minnesota and New York; in 1926, was president of the Minnesota State Dental Association. He was a member of the Munson Research and Clinical Club, First District Dental Society of the State of New York, New York Academy of Dentistry, Delta Sigma Delta, Delta Tau Delta, Sigma Xi, and Omicron Kappa Upsilon. Dr. Rowe is survived by his wife, a daughter, a son, and a brother.

Dr. Rowe's tragic death was a severe blow to the dental profession as well as to his many friends. He was assassinated in his office by one who was evidently a maniac. His untimely passing is deeply regretted.

GEORGE KERR THOMSON: 1870–1935

George Kerr Thomson died on May 2, 1935. He was born in Newcastle, New Brunswick, on Mar. 26, 1870, son of the late Judge of Probate Thomson. After graduating from the Philadelphia Dental College, in 1892, he was engaged in the drug business for a short time at Annapolis Royal, Nova Scotia, and later began the practice of

dentistry. He formed a partnership with the late Dr. Harding, at Yarmouth, Nova Scotia, moving to Halifax some years later. He was in active practice for forty-three years. He was appointed Dean of the Faculty of Dentistry of Dalhousie University, and Professor of Hygiene and Clinical and Preventive Dentistry, in 1925. He was a past-president of the Canadian Dental Association, Dominion Dental Council, Nova Scotia Dental Association, and Halifax branch of the Red Cross; also a member of the Nova Scotia and New Brunswick Pharmaceutical Societies. He had filled, also, the following positions: Secretary of the Maritime Dental Association; Secretary-registrar of the Nova Scotia Dental Board (twenty-five years); Professor of Crown and Bridge Work, Ceramics, Preventive and Clinical Dentistry; Chairman of the Army Dental Services of the Canadian Dental Association; Chairman of the Oral Hygiene Educational Committee of the Nova Scotia Dental Association; representative of the Canadian Government to the International Dental Congress (1914, 1926, 1931); representative of Dalhousie University to the British Empire Universities (1931); Chairman of the Committee on Dental Education of the Tenth Conference of Canadian Universities; Chairman of the Committee on Establishment of Pre-School-Age Dental Clinics of the American Association of Dental Schools.

Dr. Thomson was the author of many articles on Army Dental Service and Preventive Dentistry; major in the Canadian Army Dental Corps, and captain of the 63rd Halifax Rifles and Army Medical Corps; a member of the Church of England choir, and teacher in the Sunday School for many years. He was recognized as one of the outstanding men in Canadian dentistry. The profession has sustained a very severe loss in his passing. His very many friends deeply mourn his loss.

XIII. Executive Proceedings

A. Journal of Dental Research

The *Board of Editors* reported (a) that all proposals, in the report of the Executive and Finance Committees at the meeting of the Association in 1935, had been confirmed by the Board (*J. Den. Res.*, 1935–36, *15*, pp. 229–30); (b) that Dr. Theodor Rosebury had been unable effectively to inaugurate his duties as Editor, having ultimately resigned as of December

17, 1935; and that the Finance Committee had not materially increased the resources of the *Journal*. The ensuing embarrassments led, after active discussion, to adoption of the following resolution:

Resolved, that the affairs of the *Journal of Dental Research* be placed in the hands of a Committee to consist of H. C. Hodge, M. K. Hine, and H. B. G. Robinson; and that the Board of Editors be continued for the year 1936–37, to make further recommendations at the Fifteenth General Meeting on the future of the *Journal of Dental Research*.

B. REPORTS OF OFFICERS AND COMMITTEES

The *Council*, besides reporting routine ad-interim business, presented a list of nominees eligible for election to membership, having tabled several that were not regarded as eligible at present. [See the list of new members, page 380.]

The *Tellers* reported that ballots, for the officers, had been cast by the following members:

Aisenberg, Applebaum, Appleton, Arnim, Bear, Becks, Beust, Bier, Boucher, Bradel, Brandhorst, Brawley, Brown (H), Bryan, Bunting, Carr, Chase, Cook, Crane, Dahlberg, Driak, Dunning (W), Eaton, Eby, Friesell (F), Friesell (H), Gies, Goodsell, Gottlieb, Grohs, Grossman, Harvey, Hellman, Hess, Hill, Hine, Hodge, Hofer, Hollander, Husband, Hutchinson, Hyatt, Ivy, Karshan, Kitchin, Korkhaus, Krasnow, Krogh, Krogman, LeFevre, Lischer, Loos, McCall, McKay, Marshall, Mathis, Meisel, Merkeley, Merritt, Millberry, Miner, Moore, Myers (V), Naprstek, Neustadt, Noyes (H), Oartel, Orban, Paffenbarger, Palmer, Pichler, Pond, Preissecker, Price, Prinz, Randall (W), Rehak, Robinson, (H), Salmon, Schour, Schram, Sedwick, Skillen, Smith, Souder, Spector, Stafne, Sweeney, Szabo, Taber, Thoma, Thomson, Tylman, Van Huysen, Van Kirk, Varga, Volland, Ward, Waugh, Winter, Wolf, Wright (G), Wright (W)—103.

The *Treasurer's report*, examined by auditors, was stated by them to be correct. The income slightly exceeded the expenditures, the former amounting to $273 for the year.

The *Trustees of the Endowment Fund* reported that the Fund is now (March 11) $1170.32 ($1151.75 on March 15, 1935). They commented in part as follows:

"The Trustees wish to refer to a fact that may have escaped the general attention of the Association; namely, that all the money in the Endowment Fund has been derived from a personal gift by Dr. William J. Gies who, through waiving reimbursement for his expenses as Secretary for the years 1927–33, inclusive, made possible the building up of a fund amounting to over $1000.00 in the General Treasury of the Association, which [at the general meeting in 1933] was converted into an Endowment Fund by action of the membership. . . . It is . . . recommended that members of the Association seek to increase this Fund as the most practical demonstration of appreciation of Dr. Gies' gift."

The *Secretary*, in an account of ad-interim routine proceedings in his office during the year, stated that the only resignation of membership was that of Dr. B. W. Weinberger, which became automatically effective as of Sept. 13, 1935. He also indicated the steps taken by the Secretary, on his

own initiative, (a) to bring about affiliation of the American Division of our Association with the American Association for the Advancement of Science, and (b) to create the Sub-section on Dentistry of the A. A. A. S. under the leadership of our Association, with the coöperation of the American Dental Association, American College of Dentists, and American Association of Dental Schools. The conditions of the first dental meeting of the A. A. A. S. under these new conditions were discussed (*Science*, 1936, *83*, pp. 141–42; Feb. 7). [These matters were also given special attention at a collateral executive session of the American Division, at the conclusion of the afternoon session on March 15.]

C. MISCELLANEOUS RESOLUTIONS

Objection to presentation of reports on papers previously published. Resolved that papers presented at general meetings of the International Association for Dental Research be restricted to such as deal with original investigations that have not previously been published. (*Adopted*) [The term "published," in this resolution, does not include presentation at meetings of sections of the I. A. D. R.]

Objection to coöperation with "throw-away" dental journals. Whereas it is the consensus of opinion of our members that association, either as a contributor or as a member of the editorial staff, with proprietary publications that are distributed free of charge to the members of the dental profession—and whose chief object is the advertisement of commercial products—is undesirable; therefore be it *Resolved* that the International Association for Dental Research disapproves such association by its members and by applicants for membership in the Association. (*Adopted*)

Proposed amendment to the Constitution [to be brought to a vote at the general meeting in 1937]: Any member who, after the adoption of this amendment, contributes an article to, or acts as a member of the editorial staff of, any proprietary dental periodical, shall automatically forfeit his membership. [Presented by Stanley D. Tylman.]

D. LIST OF OFFICERS, AND NEW MEMBERS, ELECTED

a. Officers (1936–37)

HONORARY: *Vice-presidents*—R. GORDON AGNEW, Chengtu. GEOFFREY FRIEL, Johannesburg. CYRIL H. HOWKINS, London. KAREL WACHSMANN, SR., Prague. HERMANN WOLF, Vienna. JÓZSEF SZABÓ, Budapest.

ACTIVE: *President*—WILLIAM G. SKILLEN, Northwestern Univ. *President-elect*—PAUL C. KITCHIN, Ohio State Univ. *Vice-president*—THOMAS J. HILL, Western Reserve Univ. *Treasurer*—BISSELL B. PALMER, N. Y.

Polyclinic Medical School and Hosp. *Secretary*—WILLIAM J. GIES, Columbia Univ. *Trustee of the Endowment Fund* (three years)—BISSELL B. PALMER (Treasurer).

b. New members[5]

Baltimore, Md. HARRY E. KELSEY, D.D.S., F.A.C.D., Dental Staff, Johns Hopkins Hosp.

Chicago, Ill. STANLEY W. CLARK, D.D.S., M.S.D., L.L.B., F.A.C.D., Prof. of Materia Medica, Therapeutics and Anesthesia, GEORGE B. DENTON, Ph.D., Prof. of Technical Composition and History of Dentistry, LEONARD S. FOSDICK, B.S., M.S., Ph.D., Asst. Prof. of Chemistry, HAROLD L. HANSEN, Ph.D., Asst. Prof. of Chemistry, and GOTTFRED R. LUNDQUIST, D.D.S., M.S., F.A.C.D., Prof. of Oral Pathology—all of Dental School, Northwestern Univ. NEWTON G. THOMAS, B.A., M.A., D.D.S., 55 E. Washington St.

Cincinnati, Ohio. HUGH W. MACMILLAN, D.D.S., M.D., F.A.C.D., 1005 Union Central Bldg.

Cleveland, Ohio. FRANCIS A. ARNOLD, B.S., D.D.S., Dental Interne (U. S. Public Health), U. S. Marine Hosp. PHILANDER B. TAYLOR, A.B., A.M., Instr. in Basic Dental Technology, and CHARLES J. VOSMIK, D.D.S., Asst. Prof. of Orthodontia, Dental School, Western Reserve Univ.

Columbus, Ohio. LINDEN F. EDWARDS, B.A., M.S., Ph.D., Assoc. Prof. of Anatomy, and HARRY M. SEMANS, M.A., D.D.S., F.A.C.D., Dean, Dental School, Ohio State Univ. J. H. KAISER, Ph.C., B.S., D.D.S., 1616 E. Main St.

Detroit, Mich. SAMUEL J. LEWIS, D.D.S., Prof. of Orthodontia, Dental School, Univ. of Detroit. PERCY C. LOWERY, D.D.S., F.A.C.D., Chairman, Research Commission, Amer. Den. Assoc., Lowery Bldg.

England. Liverpool—GEORGE G. MACPHEE, M.A., M.D., Ch.B., L.D.S., Lect. on Clinical Dental Surgery, Univ. of Liverpool. *London*—ERNEST B. DOWSETT, D.S.O., M.R.C.S. (Eng.), L.R.C.P. (Lond.), L.D.S. (Eng.), Consulting Dental Surgeon, and JOSEPH L. PAYNE, O.B.E., M.R.C.S., L.R.C.P., L.D.S. (Eng.), Consulting Dental Surgeon, Guy's Hosp.

Iowa City, Iowa. LESTER B. HIGLEY, B.A., M.S., D.D.S., Asst. Prof. of Orthodontia, and ARTHUR O. KLAFFENBACH, D.D.S., Prof. of Clinical Crown and Bridge, Dental School, State Univ. of Iowa.

Lakewood, Ohio. CARL H. SCHEU, D.D.S., 14805 Detroit Ave.

[5] A list of the members, as of December 1, 1934, was published in the *J. Den. Res.*, 1934, *14*, 471; Dec. A list of the members elected in 1935 was published in the *J. Den. Res.*, 1935–36, *15*, 230; June-Aug.

Lexington, Ky. JOHN B. JUETT, B.S., D.D.S., Fayette Bank Bldg.

New York City. FRANK E. BEUBE, D.D.S., L.D.S., Asst. in Dentistry, and HERBERT F. SILVERS, B.S., D.D.S., Voluntary Clinical Asst., Dental School, Columbia Univ. WALTER A. BOSSERT, D.D.S., Dentist, Metropolitan Life Insurance Co. SIDNEY SORRIN, D.D.S., Asst. Prof. of Periodontia, Dental School, N. Y. Univ.

New Zealand. Wanganui—ROBERT E. T. HEWAT, B.D.S., D.D.S., External Examiner for Dental Division, Department of Public Health, Government of New Zealand. *Wellington*—R. M. S. TAYLOR, B.D.S., D.D.S., First Asst. Instr., Government Dental Clinic and Training School for Dental Nurses.

Peoria, Ill. R. C. WILLETT, D.M.D., 535 Jefferson Bldg.

Pittsburgh, Pa. THOMAS F. MCBRIDE, D.D.S., Instr. in Clinical Dentistry, Dental School, Univ. of Pittsburgh.

Richmond, Va. ARTHUR P. LITTLE, D.D.S., Prof. of Prosthetic Dentistry, School of Dentistry, Medical College of Va.

Rochester, N. Y. J. FRANK HALL, B.S., D.D.S., Rockefeller Fellow in Dentistry, MARION W. MCCREA, D.D.S., Rockefeller Fellow in Dentistry, and RICHARD S. MANLY, B.S., M.A., Fellow in Biochemistry, School of Medicine and Dentistry, Univ. of Rochester. PAUL LALONDE, D.D.S., 277 Alexander St.

St. Louis, Mo. EWING P. BRADY, D.D.S., Prof. of Biochemistry, Therapeutics, and Oral Pathology, Dental School, Washington Univ. L. R. MAIN, D.D.S., Prof. of Radiologic Technic and Diagnosis, Dental School, St. Louis Univ.

San Francisco, Calif. ALEXANDER J. KER, D.D.S., Instr. in Operative Dentistry, and FREDERICK W. SCHUBERT, D.D.S., Asst. Prof. of Orthodontics, Dental School, Univ. of California. ARTHUR R. MCDOWELL, D.D.S., F.A.C.D., Dean, School of Dentistry, College of Physicians and Surgeons. MAURICE L. TAINTER, A.M., M.D., Prof. of Pharmacology, Medical School, Stanford Univ.

Washington, D. C. JOSEPH L. BERNIER, D.D.S., M.S., First Lieutenant, Walter Reed Hosp. B. EDWIN ERIKSON, D.D.S., A.B., 3726 Connecticut Ave. LUZERNE G. JORDAN, D.D.S., Prof. of Prosthetics, Dental School, Georgetown Univ. STERLING V. MEAD, B.S., D.D.S., M.S., 1149 Sixteenth St., N.W. IRL C. SCHOONOVER, A.B., M.A., Ph.D., Asst. Chemist, National Bureau of Standards.

Total number of new members, 47.

Total active membership (July 31, 1936), 421.

E. MEMBERS PRESENT AT ONE OR MORE SESSIONS

SECTIONS. *Ann Arbor:* Bunting, Goodsell, Jay, Kingery, Moore, Rickert, Vedder, Ward. *Baltimore:* Aisenberg, Robinson (J). *Boston:* Boyle, Miner, Morse (F), Trimble. *Chengtu:* Lindsay. *Chicago:* Black, Blayney, Dahlberg, Denton, Fink, Fosdick, Freeman, Hatton, Kronfeld, Logan, Noyes (F), Noyes (H), Schour, Skillen, Skinner, Tylman. *Cleveland:* Arnold, Chase, Hill. *Columbus:* Cottrell, Kitchin, MacMillan, Semans. *Iowa City:* Bryan. *Louisville:* Beust, Myers, O'Rourke, Randall (W). *Minnesota:* Armstrong, Brekhus, Stafne. *New Haven:* None. *New York:* Crawford, Darlington, Gies, Stanton, Waugh, Ziskin. *Philadelphia:* Cook, Turner. *Pittsburgh:* Friesell (F), McBride, Oartel, Swanson, Van Kirk. *Richmond:* Bear, Bradel, Lyons, Van Huysen. *Rochester:* Hall, Hine, Hodge, LeFevre, Manly, McCrea, Robinson (H). *St. Louis:* Lischer. *San Francisco:* McDowell, Millberry. *Washington:* Messner, Paffenbarger, Souder. *No section:* Lowery (Detroit). *Total*—77. [All U. S. sections excepting New Haven represented in attendance.]